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PUBLIC HEALTH IN AUSTRALIA.

PART II.

THE SECOND PERIOD, 1830 TO 1850.

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"They have there neither endemics nor epidemics."—Cunningham ("Two Years in New South Wales").

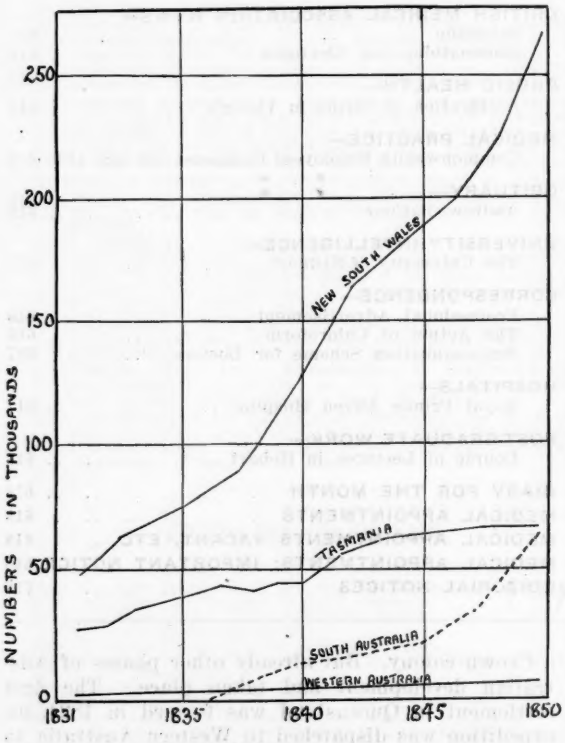
THE periods which have been selected are those which present themselves almost automatically. The original colony of New South Wales had become by 1830 a self-conscious and self-reliant community of free men with a form of constitutional government. Tasmania, although still in the early stages of its development, had a measure of self government as a Crown colony. But already other phases of Australian development had taken place. The first settlement in Queensland was formed in 1824, an expedition was dispatched to Western Australia to take possession in 1825, and an attempt at colonizing Victoria had been made in 1826. These early efforts were not very successful, but permanent colonies were established, in Western Australia in 1829, in Victoria in 1835, and in South Australia in 1836. The settlement of Queensland proceeded slowly; Victoria and Queensland were districts under the government of New South Wales until 1850 and 1859 respectively. In those years they became independent colonies.

Historical Outlines.

In very brief outline the history of each colony may be reviewed.

New South Wales.

The development of New South Wales during these years was that of steady pastoral extension with, also, the beginnings of commercial and industrial activities. The transportation of convicts ceased in 1840 and the numbers of immigrants steadily increased, so that by the end of 1850 almost the whole of the population of 266,900 was composed of other than convict stock. The increase in population, as shown by Graph I, was rapid, but uniform. This population was spread widely over the State, although the tendency to congregate in the cities had already appeared, about 22% of the population of New South Wales being, at the census of 1851, located in Sydney. The development of the community as a self-governing community had been progressive.



Graph showing the population of the different colonies from 1831 to 1850. Victoria and Queensland were at this period portions of New South Wales.

Victoria.

Founded in 1835, Victoria had had a slow progress as an entirely pastoral community. There has never (at least since the brief attempt of 1803) been a convict establishment. The population increased during the period from the small number of people who first landed, to 77,000 at the census of 1851. This colony was at first governed from Sydney as a district of New South Wales, but after a period of discontent and unrest became an independent colony in 1850.

Queensland.

The first convict settlement in Queensland was established in 1824 and from then until 1837 there was no other settlement. In 1839 only a few convicts remained and the district was thrown open for settlement. Settlement, chiefly pastoral in character, proceeded slowly; the population in 1846 was 2,258 and in 1851 it had increased to 8,575.

South Australia.

South Australia was founded as a definite emigration settlement in 1836 and exhibited very slow development along pastoral lines. The population increased from 546 in 1836 to 63,700 in 1850. There was never a convict establishment and during this period no industrial development.

Western Australia.

In 1829 a settlement of 1,003 persons was established on the Swan River. Development, pastoral and agricultural, proceeded slowly and by the first census of 1848 the population had only reached 4,622. In 1850 convict transportation was commenced and the character of the community changed. The transportation of convicts ceased in 1868.

Tasmania.

Following on a military occupation post in Tasmania in 1803, a convict settlement was established in 1804. The settlement continued for some years as a penal colony with some free settlers under the administration of lieutenant-governors responsible to Sydney. In 1825 it was proclaimed a separate colony and a legislative council appointed. With the cessation of transportation of convicts to New South Wales by 1840 thousands of convicts were poured into Tasmania and resulted in a cessation of free immigration, whilst the glut of convict labour drove free workmen from the country.

Population.

The following table (Table I) shows the population in each of the States during this period. Victoria and Queensland, being at this time part of New South Wales, are not shown separately. The

TABLE I.[1]

Year.	New South Wales.	South Australia.	Western Australia.	Tasmania.	Australia
1831	48,000	—	1,341	26,640	75,981
1832	53,524	—	1,510	28,903	83,937
1833	62,112	—	1,655	34,328	98,095
1834	66,068	—	1,800	37,688	105,556
1835	71,304	—	1,878	40,172	113,354
1836	78,929	546	1,956	43,689	125,120
1837	86,482	3,273	2,035	42,698	134,488
1838	98,176	6,000	1,928	45,764	151,868
1839	113,437	10,315	2,154	44,033	169,939
1840	127,468	14,630	2,311	45,999	190,408
1841	145,303	15,485	2,760	57,420	220,968
1842	162,317	16,340	3,476	58,851	240,984
1843	169,135	17,196	3,853	60,664	250,848
1844	178,460	18,999	4,350	62,478	264,287
1845	187,918	22,460	4,479	64,291	279,148
1846	196,704	25,893	4,547	66,105	293,249
1847	205,009	31,153	4,717	67,918	308,797
1848	220,805	38,666	4,622	68,235	332,328
1849	247,260	52,904	4,645	68,553	373,362
1850	266,900	63,700	5,886	68,870	405,356

[1] "Australian Demography—Bulletin No. 43," Commonwealth Bureau of Census and Statistics, 1925.

figures for New South Wales are correspondingly excessive.

These figures show a growth of population within twenty years from 75,981 to 405,356—a truly phenomenal development. This development was the more important in that it was a spontaneous growth, except in the case of Tasmania.

During this period of rapid increase in the population there was also proceeding a transition which altered the character and composition of the population, bringing it gradually nearer to that condition which is usually found in a stable community. This can best be seen by tracing briefly the events in New South Wales. At the commencement of the settlement of Sydney the males far outnumbered the females. This excess was so great that Phillip had instructions to procure native women from the adjacent islands for the use of the male convicts. This was not done. Very soon after the commencement of the settlement, in 1797, Phillip reported that "the vast number of women for whom we have very little work, are a heavy weight upon the store of Government."[1] Up to 1850 the percentage of children in the population was much lower than in a normal community. Gipps, reporting on the population of the colony in 1845, summarized the position as follows:

> There is an estimated increase in the population during the year of 9,572 souls, bringing the population up to 173,377. It is remarkable, however, that while there has been this increase by births and immigration, there has been, as there also was in 1843, a decrease in the adult population, both male and female, which can only, I imagine, be explained by the dying off of convicts. The adult males in the colony are still more than double the females.[2]

The high death rates of 1832 to 1842 may have been due to the dying off of the old convicts who, transported between 1788 and 1800, may have been in the colony for periods up to fifty years.

Immigration.

The following table (Table II) shows the course of immigration into New South Wales.

TABLE II.

Year.	Number of Immigrants Embarked.
1825	485
1826	903
1827	715
1828	1,056
1829	2,016
1830	1,242
1831	1,561
1832	3,733
1833	4,093
1834	2,800
1835	1,860
1836	3,124
1837	5,054
1838	14,021
1839	15,786
1840	15,850
1841	32,625
1842	8,534
1843	3,478
1844	2,229
1845	830
1846	2,347
1847	4,949
1848	23,904

It will be seen on comparing this table with the computed death rates that the high death rate was falling sharply when the great wave of immigration occurred between 1838 and 1841, and that, although this great ingress of newcomers coincided with the financial depression and the drought of 1840-1841, and was a factor in the unemployment then rampant, the death rate was not raised.

Birth Rate.

Births were not registered, but baptisms were recorded. From these a crude kind of birth rate per thousand of population has been compiled. This rate, for each year shown, is as follows:

1837	26·4	1845	36·4
1838	28·2	1846	37·1
1839	28·2	1847	34·4
1840	30·1	1848	35·6
1841	—	1849	31·4
1842	31·3	1850	30·2
1843	32·4	1851	27·3
1844	34·0		

An error in these figures is towards a lower birth rate than the actual. These birth rates are high compared with those in a stable population.

There were thus three movements taking place—a high death rate among the older adults, a considerable immigration of healthy adults, presumably for the most part young, and a high birth rate. These all were making for the stabilization of the population on a normal composition.

The Records of Disease and the Course of Mortality.

New South Wales.

The picture that Cunningham drew of health conditions in New South Wales seems to be generally true of the period now under review. It will be shown that some modifications are necessary. For this period causes of deaths were not recorded, but approximate totals of actual deaths (in reality of burials) are available. From these is compiled the following table[3] (Table III).

TABLE III.

Year.	Deaths.	Crude Death-Rate per 1,000 Living.
1829	615	15·0
1830	570	12·8
1831	582	12·1
1832	880	16·4
1833	1,150	18·5
1834	1,164	17·6
1835	1,453	20·4
1836	1,628	20·6
1837	1,799	20·8
1838	2,104	21·4
1839	2,481	21·9
1840	2,382	18·7
1841	2,400	16·5
1842	2,280	14·4
1843	2,293	13·5
1844	1,899	10·6
1845	1,787	9·5
1846	2,190	11·1
1847	2,302	11·2
1848	2,103	9·5
1849	2,703	10·9
1850	2,585	9·7

The high mortality rate which appears to have prevailed amongst the general population in New

South Wales during the period 1832 to 1842, does not seem to have occasioned contemporary comment, nor did the lower rate during the years 1843 to 1849, nor the subsequent rise again after 1851. In the absence of definite records it is impossible to find in the mortality curve for the period the effect of epidemic diseases. An analysis of the census figures and of the mortality of the sexes and of adults and children indicates that children and female adults died at a somewhat higher rate than the adult males, but generally this analysis rather supports the hypothesis that the trend of the mortality curve in the period 1830 to 1850 was dependent on factors involved in the stabilization of the population rather than on the incidence of epidemic disease. It is unfortunate that during this early period of stabilization of the population so little information is available as to the incidence of the particular diseases, beyond the statements that dysentery was a very prevalent and fatal disease.[4] From the evidence available it appears that neither typhus nor typhoid fever was prevalent, Asiatic cholera had not appeared, but that diarrhœal conditions were present and were confused by some practitioners with true Asiatic cholera; influenza was epidemic on two occasions, scarlet fever had been introduced, small pox had recurred, and there is a doubtful reference to measles. In the confusion of clinical ideas of that time and the absence of records of clinical observations, it is not possible to deduce more than this general conception of the position as regards the incidence of individual diseases during this period. It may be conjectured from the records that probably typhus fever had been introduced, but had never become endemic, and in regard to typhoid fever, that cases were occurring, but that epidemicity had not then been reached. It is, however, noteworthy that by 1850 overcrowding was commencing in the metropolis, with the resultant insanitary condition of slum areas—a condition which was more fully emphasized at a later date in the minutes of evidence of the commission appointed in 1860 to inquire into the condition of the working classes of the metropolis.

Leprosy.—Leprosy, according to Watson,[5] first appeared in the Sydney Hospital records of 1838 as lepriasis, and in 1841 as lepra, but Thompson[6] accepts this entry in the records of a later year (1849) as indicating *lepra vulgaris* (psoriasis) and dates the development of the first case of leprosy in New South Wales as 1859.

Victoria.

On the evidence of Thomas[7] and Clutterbuck[8] the most prevalent disease in Victoria prior to 1850 was dysentery. "It attacked people of all ages and was extremely fatal amongst infants. It was asthenic and never malignant." It caused great mortality in 1840. "Fever, dysentery, inflammation of the liver, rheumatism, apoplexy, palsy, croup" were "of as frequent occurrence as in England." Typhoid fever (called "low fever") is said to have been rife in the neighbourhood of Melbourne in 1841. Of tuberculosis Clutterbuck writes:

> Consumption, that greatest scourge to persons residing in England, consigns many victims in Port Phillip to a premature grave. To imagine, as many do, that emigration to this colony tends either to prevent the development of this complaint, or to arrest its progress, is a fatal delusion . . . the most intense cold of England does not produce so baneful an influence on a habit predisposed to consumption as the sudden vicissitudes of the Australian climate.

Queensland.

In 1829 the settlement at Moreton Bay (now Brisbane) was moved from Redcliff to the present site of Brisbane, as the water was bad and sickness common. In 1828 there were admitted into hospital 956 patients. Of these 27 were sufferers from "intermittent fever" and 161 are recorded under the name of *febris*, of whom none died. These cases were probably not all intermittent fever. This term at that time would be likely to include many cases of febricula, associated with its ordinary causes, sore throats, catarrhal diseases *et cetera*, as well as some cases of typhoid and possibly also of dengue. There were 665 patients with intermittent fever recorded as being under treatment for the whole year 1832. Of these four died. There is nothing in the records to indicate whether these patients came from other districts that were known to be malarial. The principal disorders of the people in this settlement for many years were intermittent fever, dysentery and ophthalmia.

In 1845, it is recorded, the common diseases of the colony were ague, continued fever, chronic rheumatism and influenza, the first two being caused by exhalations of vegetable miasma, the next by undue exposure to wet and night air, the last by some uncommon state of the atmosphere, producing at first ordinary colds, which soon became infectious and epidemic.

Jackson[9] considers that typhoid did not then occur, at least among the hospital patients.

South Australia.

Although the colony of South Australia was founded in the year 1836, there is not available much authentic information concerning the occurrence of disease for the first twenty years of its history. Wilkinson, writing in 1848,[10] stated that:

> The general healthfulness of the place gives very little scope for medical information. Many people are troubled with dysentery, which, if taken in time, is in general easily subdued and seldom produces fatal results.

Western Australia.

Fairly complete records are available of the conditions in Western Australia during the period between 1830 and 1850. In 1831 a local surgeon refers to the only diseases likely to affect the settlement as being dysentery and ophthalmia. In 1836 a descriptive account of the conditions in this colony was written by Milligan,[11] who gives the following information:

> Cases of dysentery appear to occur throughout the year, but it prevailed in April, 1830. The disease appears to have been of a mild nature, terminating favourably in a

few days. Scurvy first manifested itself in June, 1830. Of 63 cases treated, 57 recovered and six died. In May, 1830, a few cases of continued fever appeared. A mild form of cholera showed itself in October, 1832, but the disease bore no resemblance in severity to Indian cholera and readily subsided under treatment.

Thereafter, until 1850, the record is one in which dysentery, typhus and continued fever (both of which were probably enteric fever) were responsible for a great deal of the sickness and many of the deaths. A full account is to be found in "The History of Intestinal Infections in Australia." "Continued fever," which may be accepted as enteric fever, was unduly prevalent in the summer of 1844-1845.

In 1845 influenza was prevalent amongst the white inhabitants, but was not dangerous.

There is a suggestion in Milligan's account that some vessels had arrived from England between 1829 and 1836 on which typhus had prevailed during the voyage.[12] The Registrar-General's report for 1843 contains the statement that small pox, measles, whooping cough, typhus or puerperal fevers were unknown in the colony.

The following is the number of annual deaths so far as these are known:

1837	21	1844	53
1838	36	1845	63
1839	24	1846	39
1840	20	1847	69
1841	N.A.	1848	68
1842	29	1849	29
1843	41		

Tasmania.

After 1824 there were more complete records in Tasmania and from 1824 to 1835 admissions to the hospitals were tabulated according to the nature of the disease. The intestinal conditions—cholera, colici, diarrhœa, dysenteria, enteritis and febris—all occupied a prominent place. In the years 1834 and 1835 these were specially prevalent. Again, in 1839-1840 there was an epidemic of "fever" sufficiently great to have given rise to considerable comment.

There was a considerable amount of scurvy among the convicts at Port Arthur between 1831 and 1835.

In the years 1839 and 1840 there was a very severe outbreak of typhus fever in Hobart. This epidemic appears to have commenced in July, 1839, being, according to Hall,[13] definitely associated with the arrival, in that month, of the prison ship *Marquis of Hastings,* with 240 male convicts. Very little fever had existed up to that date in Hobart generally. Between August, 1839, and July, 1840, there were recorded 914 cases, amongst which there were 82 deaths. A notable feature of this outbreak is that free persons, as distinguished from convicts, escaped almost entirely. It is recorded that "overcrowding was always practised in the prison stations; the men being lodged in small bunks, one tier above another, with sleeping places of about 108 cubic feet."

There is no definite information as to the course of these fevers between 1840 and 1844, but there is a contemporary account of the type of fever which prevailed immediately prior to 1843. Power[14] described it thus:

> The form which it generally assumes, though partaking very much of that of typhus, presents some features which distinguish it from most other fevers, and on account of these it has received the name of "colonial fever."

The description which follows is that of enteric fever.[15] Cases of "fever" occurred in considerable numbers up to 1850. Hall[13] states that:

> A large proportion of the deaths from fever were from immigrants recently arrived in the Colony. Emigrant and convict ships have often brought to the Colony severe types of fever, but beyond those immediately in contact with the sick in the hospitals and prisons, the diseases have not spread epidemically. Our climate seems antagonistic to the spread of febrile disease, when the people enjoy ordinary domestic advantages. Fever occurs where the local defects of sewerage, accumulations of filth *et cetera* can engender it.

It is reasonable to deduce, from the evidence elsewhere assembled, that about 1839 to 1841 there was a very severe epidemic of typhus fever amongst the convicts, introduced by a convict ship and spread through overcrowding and insanitation. Later there became established endemic enteric fever, which was not limited to the convict population and was associated with insanitation.

Course of Some Diseases.

A general survey of the conditions in each of the colonies having now been given, it is possible to indicate briefly the course of some of the diseases.

Croup.

Croup is recorded in Western Australia and Tasmania before 1850, but only in small numbers. On the authority of Clutterbuck it occurred also in Victoria. The identity of this condition is uncertain. At some time between 1827 and 1842 croup was first recognized in Australia. It remained present throughout succeeding years, causing deaths, with some fluctuations in annual incidence, until the dramatic appearance, in 1858, of the "new disease" which was recognized as diphtheria.

Influenza.

Influenza was reported in Sydney in 1836 and 1838. In the latter year a very severe epidemic occurred in October and November. In 1838 it occurred also in Tasmania. It occurred in later years as follow: South Australia, 1839 and 1844; in Tasmania, South Australia and Victoria in 1847, 1848, 1849; and in Victoria and Tasmania in 1851, 1852 and 1853. The epidemics of influenza in England in 1837 and 1847 were sufficiently dramatic and severe to be described as "explosions," reappearing, as they did, after a long period of quiescence in England.

Hirsch speaks of influenza thus: ". . . in truly pandemic form we meet with authentic influenzas in the years . . . 1836-37, 1847-48, 1850-51."

This occurrence of influenza introduces the student to the first definite occasion on which Australia shared in a general epidemic manifesta-

tion of the disease in several countries. It was also the first occasion on which several States were simultaneously affected with an epidemic disease. The coincidence in respect of time was not, however, exact. The delays in transit, the infrequency of communication, postponed the epidemic in mainland States in relation to Europe, and in Tasmania in relation to mainland States.

Whooping Cough.

It has been recorded that whooping cough was introduced into Sydney in 1828. It was very prevalent in Tasmania in 1833 and was from there introduced into Western Australia by a military detachment. It apparently became established after that date and was especially prevalent in Tasmania in 1838 and 1842, in Western Australia in 1848. It is recorded in 1848 as having been endemic in South Australia and, it may be inferred, was well known in Victoria by 1849.

In 1837 and 1838 two vessels were quarantined at Sydney for whooping cough; and on one vessel, quarantined at Melbourne in 1849, whooping cough had occurred during the voyage.

Scarlet Fever.

From the available sources of information it may be assumed that scarlet fever was not recorded in Australia before 1833, when a definite case was observed in Tasmania. The first cases in Victoria and New South Wales were recorded in 1841. It does not appear to have been a common complaint (if it occurred at all) in South Australia in 1848, but it was established in that State by 1859. It was not recorded in Queensland before 1858 nor in Western Australia before 1865. In Tasmania there is an absence of information after 1843, but it is recorded that "no part of the island escaped its visitation in 1843." There was again an interval of ten years, during which the records are unavailable, but in 1853 "another epidemic visitation scourged the whole colony," 230 deaths occurring from this disease in Hobart alone. Deaths occurred in Tasmania in four of the five years 1857 to 1861, 1860 escaping.

In Victoria, after its initial appearance in 1841, it remained present in the colony, assuming, in 1848, a malignant type, the attacks being very general, and in a great many cases very severe and rapidly fatal.

In 1837, 1838 and 1849 one vessel each year arrived at Sydney and reported the occurrence of scarlet fever during the voyage.

Measles.

Apart from a suggestion which has not been verified, that measles occurred at "Illowalla," New South Wales, about 1832[16] and the record of two deaths in Hobart from measles in 1842,[17] the records indicate that measles was introduced into Australia at Melbourne in 1850 by the ship *Persian*. The disease seems to have reached New South Wales in 1853, Tasmania in 1854, Queensland in 1857 and Western Australia in 1860. It had become established in South Australia by 1859.

One vessel, the *America*, was quarantined at Sydney in 1829, having had a smart outbreak of measles during the voyage. Other vessels were quarantined on arrival at Sydney, one in 1838 and one in 1849. In 1850 one convict ship was quarantined at Fremantle, having had a considerable number of cases of measles on board during the voyage.

Puerperal Fever.

There is an interesting record relating to puerperal fever. Dempster, writing in 1835 of Tasmania, said: "Puerperal fever showed itself (I think for the first time) in the autumn of 1833." It will be remembered that this was the first year in which scarlet fever was recorded.

Small Pox.

Reference has already been made to the small pox outbreak among the aborigines in the interior districts. This outbreak "appeared not to be an introduced one,[18] and it extended far inland. There can be little doubt that it extended into Queensland and along the Murray River into South Australia, being actually observed amongst the natives by Sturt. It caused a very high mortality amongst the aboriginal tribes."

There are also fleeting glimpses to be had of an epidemic introduced into the Northern Territory at some time during the decade 1860-1869, probably in the earlier years. It affected only the aborigines.

During this period there were quarantined at Sydney for small pox the *Bussorah Merchant* (1828), *Mermaid* (1830), *Prince Regent* (1833), *Canton* (1835) and, possibly also, *Pitt* (1792) and *Bussorah Merchant* (1837). Probably, also, there were vessels quarantined in Tasmania, but of these no record has yet been found.

A vaccination institute was officially established in Sydney in 1847.

Quarantine.

In 1832 there was passed the first statute enacted by an Australian legislature which had any relation to public health. This was the *Quarantine Act* of July 28, 1832. The form of this act, as might have been expected, was copied almost *verbatim* from the *Quarantine Act* of England of 1825. One important difference was that while the English act recited that it was "probable that the plague or other infectious disease or distemper highly dangerous to the health of His Majesty's subjects" might be introduced, the New South Wales act substituted "malignant cholera" for "plague" in the above phrase.

In 1841 this act was amended to give power to the health officers to order a vessel to perform quarantine without awaiting the Governor's authority; and also it extended the executive provisions to include the newly founded settlement of Victoria.

In 1849 the act was further amended to include all ports in New South Wales and the police magistrate was given authority to order a vessel to perform quarantine.

In 1834 Governor Bourke was required to inform the Secretary of State for the Colonies as to the procedure adopted under the *Quarantine Act*. Bourke replied that a proclamation had been issued on August 14, 1832 (repealed July 1, 1834) proclaiming the United Kingdom as infected with cholera and requiring all vessels therefrom to await inspection on arrival. If any case of cholera had occurred on the vessel within thirty days of arrival, the vessel was to be ordered to perform quarantine according to the regulations.[19]

In 1852 a select committee was appointed in New South Wales to inquire into the quarantine laws, and particularly the proposed modifications in these laws. After this committee had reported, an amending *Quarantine Act* was passed in 1853. This act provided for administrative detail in several directions, such as requiring the master and the dispenser to answer all questions concerning the health of the crew and passengers, prescribing a quarantine line, prohibiting communication with the shore until after the vessel had been passed by the health officer, and exempting vessels from the other Australasian colonies and New Zealand from medical inspection so long as those places were not proclaimed as infected.

Thus began the Australian quarantine system. This beginning contains the first indication of progressive modification of an English law, originally automatically adopted, to meet local needs and conditions. This sequence is to be found repeatedly in the history of administration in Australia.

Certain other acts were passed during this period.

In New South Wales an act to prevent the adulteration of bread, meal and flour was passed in 1835. An act to define the qualifications of medical witnesses at coroners' inquests was passed in 1838; a *Census Act* and a *Lunacy Act* were passed in 1845; and in 1850 was passed an act "for the sewerage, cleansing and draining the city of Sydney and to promote the health of the inhabitants thereof."

In other States other acts relating to public health were passed.

Thus were introduced the beginnings of an orderly public health system in Australia, beginnings which were embryonic enough, but which were destined to grow steadily into a complete legal and administrative code.

Review of the Second Period.

During this period Australia began its career as a continental nation. All the States were established as colonies and the population increased rapidly. The birth rate was high, following on the great immigration which had taken place. In the early part of the period the death rate in New South Wales was high, due probably to the deaths of large numbers of the convicts transported between 1788 and 1800. During this period also there was proceeding a change in the age constitution of the population, bringing it to a closer approximation to the age constitution of stable populations.

Typhus fever had declared itself severely under the conditions of gross overcrowding and insanitation in the convict establishments of Tasmania; but, as these predisposing conditions were not present in the convict stations in Western Australia, typhus did not occur there.

Influenza was prevalent on two occasions during the period; whooping cough and mumps were introduced and were very severe, but did not persist. Dysentery was the characteristic disease in each of the settlements on their first establishment. Tuberculosis of the lungs was known and several writers concur in the statement that it showed a special activity amongst adolescents.

Small pox showed definite recurrence in epidemic form in the interior of New South Wales.

Several vessels arrived infected with various diseases, and the danger from these led to the passage of a *Quarantine Act*. This act, copied at first from the English act, passed through various amendments to adjust the legal code to local requirements and developments. The passage of various acts relating to sanitation, census, control of medical practice, lunacy, and the adulteration of food, represented the earliest stages of a public health system.

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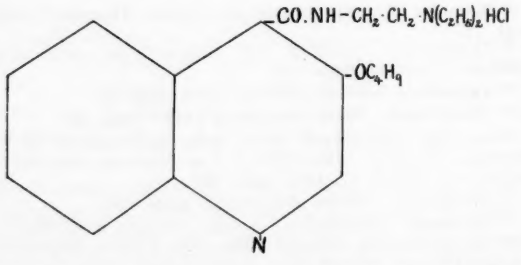
"PERCAIN": THE NEW LOCAL, REGIONAL AND SPINAL ANALGESIC OR ANÆSTHETIC.

By R. GRAHAM BROWN, F.C.S.A.,
Brisbane.

As is well known, I have for some years now been an ardent supporter of the use of local anæsthesia (analgesia) whenever possible, and I have naturally been deeply interested in several papers which have recently appeared on a new local anæsthetic, "Percain," particularly the paper by W. Howard Jones,[1] and the conjoint paper by Norman C. Lake and C. Jennings Marshall.[2] I awaited the arrival of this drug on the Australian market so that I could satisfy myself as to its qualities. During the latter part of November I was supplied with samples, and during the months of December and January and the first week of February seventy-five operations about the head and neck were performed by me in my private practice under the influence of injections of this preparation. In addition other operations were performed under the influence of local applications of the drug to mucous membranes. I am so impressed with its superiority in all but one respect, namely, ischæmia (which respect I will mention later) to all other forms of local anæsthesia that I think it is my duty to add my weight of evidence and to bring the matter before the notice of practitioners in Australia in a more intimate manner perhaps than is done in the advertising literature of the manufacturers.

Chemical Properties.

From the manufacturers we learn that "Percain" is the hydrochloride of diethyl-ethylene diamide of alpha-butyl oxycinchoninic acid and has the structural formula:



It belongs to a group of chemical combinations which until quite recently were unknown to possess anæsthetic properties. It does not belong to the same family as cocaine or "Novocain," but it is a derivative of quinoline which is related to quinine. It forms colourless crystals, odourless and tasteless; it crystallizes at 32·2° C. (90° F.) and melts at 36·1° C. (97° F.). "Percain" is readily soluble in water and alcohol, the solution having a neutral reaction. For surgical purposes it is dissolved in saline solution, when it forms a neutral solution, but if brought into contact with alkali it is thrown out of solution. In consequence it is essential to use distilled water and sodium chloride free from alkali and the solution must be kept in alkali-free glass containers. Should alkali contamination occur, it becomes evident by a cloudiness which readily disappears upon the addition of one drop, more or less, of dilute hydrochloric acid to the hundred cubic centimetres. The solution can be repeatedly sterilized by boiling without its losing any of its activity and it is miscible with adrenalin. The manufacturers recommend that solutions be prepared freshly as required and that adrenalin be added only at the time of use.

"Percain" has an intense and prolonged anæsthetic effect and laboratory experiments have shown that a solution of the drug of one in 125,000 has a very pronounced effect on the rabbit's cornea in comparison with the minimum cocaine concentration of one in 10,000, and therefore, weight for weight, it is by far the most powerful local anæsthetic known. "Percain," however, is far from being non-toxic, the relative toxicity being about 4·0 (using the British Medical Association Committee's scale of relative toxicities: Cocaine, 1·0; stovaine, 0·625; "Novocain," 0·49; β-eucaine, 0·414), but seeing the dilution in which it is used, this factor is reduced well below that of either the above mentioned drugs. It has been shown also that the anæsthesia produced with a one in 1,000 solution of "Percain" lasts one hundred and thirty minutes, but only fifty-five minutes with a solution of cocaine of the same concentration. It is surpassed in this respect by quinine and urea hydrochloride, but has not the same marked contraindications. It has been likewise shown that the intact skin of the frog (comparable in many respects, it is reported, to the mucous membranes in man) resists the passage of "Percain" into the adjacent tissues, although the sensory nerves of the skin itself are completely anæsthetized. It is claimed, therefore, that when "Percain" is used for surface anæsthesia the risk of poisoning by absorption through the mucous membrane is much less to be feared than when cocaine is employed. Experiments with injections into man have shown that a solution of "Percain" of one in 1,000 has a similar intensity of action as a solution of cocaine of one in 100, but with the added quality that the anæsthesia lasts much longer.

The injection of "Percain" solution does not cause any irritation in the tissues. Up to the present time thousands of operations performed under its influence have been reported, and amounts as much as 400 cubic centimetres of a one in 2,000 solution have been used without any apparent bad effects to the individual. The quality of prolonging anæsthesia is of great value, as in some of the large operations the patient has had post-operative freedom from pain for from six to twelve hours before the effects have worn off. Some cases have been reported in which the anæsthesia lasts as long as twenty-two hours, but five hours have been suggested as the average of complete anæsthesia, followed by a period of hypoæsthesia of the same duration. Insensibility of the tissues is apparent almost immediately after injection and this increases in intensity and extent

during the first five to ten minutes, thereafter it is complete, provided the correct technique has been employed. Injections of "Percain" are generally well tolerated, and neither lesions of the tissues nor trouble in healing of the wound have been noticed. "Percain" has a greater vaso-dilator action even than "Novocain" and so the addition of adrenalin is essential. For anæsthesia to mucous membrane a 2% solution is recommended to which adrenalin is added.

The drug is a product of the Society of Chemical Industry in Basle, Switzerland ("Ciba"), and the above is a summary of the main claims of the manufacturers.

Personal Experiences.

I am pleased to state that my experiences up to date, small though they may be, confirm the above mentioned clinical claims. I have found that for injection purposes a one in 1,500 or even one in 2,000 solution is sufficient for all my requirements, and included in my list of operations are tonsillectomies, radical antral operations, operations about the mouth, radical mastoidectomy, complete gland dissection of the neck, external operation upon the larynx, reconstruction of nose by costal cartilage implantation, and various plastic operations about the face. I have found that the anæsthesia is obtained almost immediately upon injection, and that it soon becomes profound. It is noticed, when incising the tissues a short interval after infiltrating, that they are very little (and frequently not at all) changed from the normal. I frequently block the ethmoidal nerves in the orbit and in the past very often a considerable amount of œdema of the eye lids was associated with the use of "Novocain" solution, which sometimes lasted for several days. With "Percain" injections a very definite reduction in the amount of this reaction results, more than suggesting thereby that the "Percain" solution has a less harmful effect upon the tissues. The healing reaction following the use of this drug, as compared with other anæsthetic solutions is, if anything, better, and I am convinced that the anæsthetic action is more prolonged. In dealing with patients who have been subjected to enucleation of the tonsils, this prolongation of the analgesia is a decided advantage, and not only am I convinced of this fact, but the nursing sisters in charge of my patients, who are really better able to judge, support my opinion.

With regard to anæsthesia of mucous membrane, I have, as recommended, used a 2% solution of the drug combined with a one in 5,000 adrenalin solution. Owing to its being tasteless it is much more pleasant to use, and I have found that it produces a more profound anæsthesia than a solution of 10% cocaine and one in 5,000 adrenalin. The ischæmia, however, is not so marked as when using cocaine and adrenalin. The usual procedures which one performs in one's consulting rooms can be as effectively carried out with 2% "Percain" as with 10% cocaine solutions, that is, antral puncture, incision of drum head, cauterization of nasal mucous membrane and adenoid tissue about the pharynx, and such like. I have used this strength for packing the nose, for submucous resection of the nasal septum and in combination with ethmoidal nerve blocking with a one in 2,000 solution, in such operations as intranasal work upon the frontal, ethmoidal and sphenoidal and antral sinuses, with satisfaction.

"Percain" has a definite vaso-dilator action and in consequence adrenalin must be used in combination. Even with the same amount of adrenalin as is used with "Novocain," I have found that the ischæmia is less marked, and in tonsillectomies this at first caused me some slight annoyance, being accustomed, when I used solutions of "Novocain" and adrenalin, to an almost bloodless field. What surprised me was that, whereas occasionally when I used "Novocain," the solution, when injected into the capsule, produced a mass of œdema there, but when such an event happened with "Percain" solution there was only the slightest trace of evidence of œdema. My usual procedure in operating under local anæsthesia is to prepare my patients by using suitable sedatives, mainly in the form of a barbitone derivative, preferably "Allonal" or "Sodium Amytal" and morphine, and the amounts injected for the various operations are as stated below.

Feeling confident in my methods of injection, I am now using a one in 2,000 solution, but it would be as well for the surgeon at his first series of operations under the influence of this drug, for obvious reasons, to use a solution of one in 1,500 or even a one in 1,000 and to inject slightly larger amounts. Wherever possible I use the "block" method of producing anæsthesia. For tonsillectomy I use about 7·5 cubic centimetres a side, injected at three points, which produce not only anæsthesia about the tonsil itself, but also anæsthetizes the pharynx and the base of the tongue. For ethmoidal nerve blocking in the orbit two cubic centimetres are used. For radical antral operations I use about ten to twelve cubic centimetres a side, including two cubic centimetres for ethmoidal nerve blocking. For block dissection of the neck, from 75 to 100 cubic centimetres are used. These amounts are the same as when using a 1% "Novocain" and adrenalin solution.

I feel that there are several reasons why this drug should replace cocaine, particularly in its surgical applications. When a solution of one in 2,000 is used, one realizes that the toxic element must be reduced to almost vanishing point, and considering that the drug manifests all the qualities which have been claimed for it, there is very good reason, therefore, to eliminate cocaine from our list of drugs—at any rate in connexion with operative work. On referring to the price of cocaine, one finds that a high grade quality cannot be obtained in Australia today for less than £1 7s. 6d. an ounce. A five-gramme bottle of "Percain" costs seven shillings, which works out at about £2 2s. per ounce, and as only a 2% solution of "Percain" is required for

local applications to mucous membranes in place of a 10% solution of cocaine, the cost is about one-third or even one-quarter that of cocaine. "Percain" is tasteless, which should be a point in favour of its use, at least in private practice.

It is never advisable to have solutions of cocaine lying about, and it is a well known fact that many ounces of this drug are wasted *per annum* in public hospitals, with the corresponding financial loss, because of the deterioration and contamination which may take place in these solutions when left standing for any length of time.

Cocaine is a dangerous drug and, considering the risk by possible mistakes and also by the increased facility for obtaining the drug by cocaine addicts, it is possible that the appearance of this new drug, "Percain," may go a long way towards practically eliminating cocaine from medical use. I feel that from this point of view, and seeing that we can obtain a more efficient effect with the use of "Percain," the question of its routine use should be seriously considered by authorities and by individuals.

Contrasting "Percain" with "Novocain," its anæsthetic effects are considerably more pronounced for local application and for injections I feel that it is preferable to "Novocain" in many respects, and the cost is considerably below that of the latter drug.

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THE GONIN OPERATION FOR THE CURE OF IDIOPATHIC RETINAL DETACHMENT.

By J. BRUCE HAMILTON, M.B., Ch.M. (Sydney), D.O.M.S. (London), D.O. (Oxon),
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STALLARD,(1) in *The British Journal of Ophthalmology* of January, 1930, wrote on the "Causes and Treatment of Simple Detachment of the Retina" and in a most interesting manner compared the results of the treatment of 100 patients by various methods. From my experience the numbers of recoveries he reported from each of these methods were exceptionally high. Further, he described in some detail the many operative procedures undertaken for the cure of this very distressing malady since 1860 and ended this section with a brief allusion to the recent work of J. Gonin, of Lausanne. The reader of this article by Stallard will at once realize from the multitude of methods, both operative and non-operative, used for the attempted cure of simple retinal detachment, and from the recoveries reported as results of each method, that the outlook is not bright.

Amongst the discussions at the thirteenth International Congress of Ophthalmology, held at Amsterdam in September, 1929, was one on simple retinal detachment, opened by W. Kapuscinski (Posnan), H. Arruga (Barcelona), J. Gonin (Lausanne) and Periz-Bufill (Barcelona). Gonin,(2) in the course of his remarks, referred so disparagingly to the work of British ophthalmologists in the treatment of this condition that, shortly after the congress closed, several English oculists journeyed to Switzerland to investigate the technique practised at Lausanne. So well were they received and so favourably impressed with the results witnessed that, on their return to England, an immediate start was made to employ the Gonin technique. At that time I had the good fortune to be on the house staff at Moorfields Eye Hospital, where the first attempts were made to carry out the technique in detail. So favourably impressed were we with the results obtained at Moorfields that we had treated over sixty patients by this method up to August, 1930. As it is felt that not temporary, but permanent, restoration of the field of vision is to be aimed at, no report on these and future cases will be issued till the middle of the present year. In the midst of our work at Moorfields, Gonin visited England and, at the Oxford Ophthalmological Congress in July, 1930, further elucidated his methods and enlightened us on several baffling points. His remarks, in perfect English, were a great delight to the large and distinguished gathering.

The ætiology and cure of simple retinal detachment have been the life work of Gonin and one felt as one heard him talk so fascinatingly of his subject that the ætiological factor was to him much more important than the cure he has evolved. Yet to the world at large he has become famous only for his remarkably successful cures.

Ormond,(3) in *The British Medical Journal* of May, 1930, discusses in some detail Gonin's theory as to the cause of simple retinal detachment. The reader is referred to his interesting article. Here, suffice it to say that as the cause of every detachment (in the remaining portion of this paper a simple detachment of the retina is referred to as a "detachment") there is a hole (or holes) in the retina through which the fluid portion of the vitreus passes into the subretinal space, floating the retina off the choroid. If the hole or holes are blocked, the retina returns into position and permanent restoration of function results.

From the outset of our work at Moorfields three outstanding problems presented themselves, namely: (i) The location of the holes in the retina; (ii) the question as to whether all types of holes are suitable for operative treatment; (iii) the acquisition of a perfect operative technique, which appeared to be essential to the success of the operation.

I shall now discuss the treatment of detachments, paying special attention to the three problems above mentioned.

Finding the Site of Detachment.

I do not feel that there is any difficulty in finding the detachment, as usually there is a fairly large

one before the patient notices any deterioration of vision, but I do admit that sometimes after operation there is difficulty in saying whether the detachment has been completely cured or not. The perimeter will always decide this question.

Finding the Aperture or Apertures.

Searching for the hole or holes in the detachment is truly a wearisome business and may occupy many hours spread over several days. In *The British Journal of Ophthalmology* of September, 1930, I[4] described in detail the intricacies of this exhausting pastime and divided detachments into two groups for hole-finding:

1. Those in which I did not expect to find a hole; this group included sarcomata and inflammatory detachments.

2. Those in which I expected to find one or more holes. This group included the simple detachments that occur in myopic eyes and in emmetropic eyes after local or general trauma. In this class in at least 90% of instances one or more holes are present. In my article, above referred to, I gave many hints for the localizing of them and stressed the fallacies encountered. There I stated that I had never seen a hole behind the equator of the globe, but since writing that article I have seen one hole at the macula itself—a position quite inaccessible. Further, I described the three main types of hole found, namely: (*a*) punched-out hole with pigmented edge, (*b*) horseshoe shaped rent, (*c*) disinsertion of the retina at the *ora serrata*. I would now refer the reader to the recently published *Transactions of the Thirteenth International Congress of Ophthalmology*. In Volume II, facing page 658, are excellent illustrations of these various types, by Arruga.[5]

Suitable Type of Hole for Treatment.

The most suitable hole or holes are those which can be blocked by one "ignipuncture"; the necessity of a second operation decidedly mitigates the chances of success. Gonin states that he finds disinsertions at the *ora serrata* sometimes difficult, for quite often after one disinsertion has been blocked, the retina becomes detached from another spot at the ora. At Moorfields we found some of the larger horseshoe shaped rents particularly difficult to occlude, and we eventually decided not to operate if these were extensive, as the results were so poor. Further, we refrained from attempting to deal with holes at the macula, although an instrument has been devised for this particular purpose.

Localizing the Hole in Relation to the Exterior.

Having found the hole or holes, the next step is to localize it in its relation to the outer wall of the globe. Both Juler,[6] in *The British Journal of Ophthalmology* of February, 1930, and Ormond, in his article already referred to, describe the procedure in some detail. If the patient's eye be fully rotated, the most anterior part of the retina that is visible may be considered to be the *ora serrata*, and we know that the ora is situated 8·5 millimetres behind the limbus of the cornea. Now the diameter of a normal optic disc is 1·5 millimetres, so, having accurately judged the diameter of the patient's disc, the distance from the *ora serrata* to the hole is estimated in disc diameters which are then converted into millimetres. For example, if the hole is three disc diameters from the ora, then it is 4·5 millimetres from the ora and 13·0 millimetres from the limbus (Figure I).

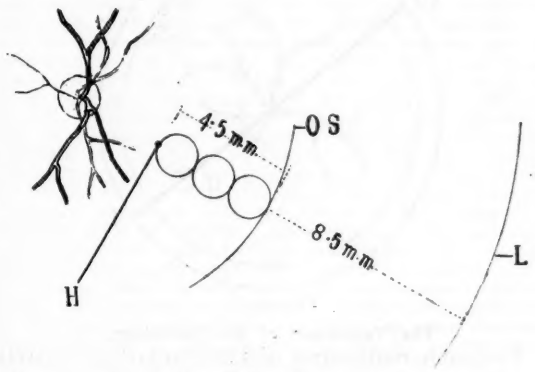

FIGURE I.

Next it is necessary to decide, in terms of the clock face, the meridian in which the hole stands.

Marking the Meridian.

For marking the meridian of the hole at the limbus a Saunders needle is required, or, preferably, a needle devised by Gonin's first assistant, Amsler, and now made by John Weiss and Sons, of London. Also required are a concentrated solution of gentian violet and a stick of solid indian ink. The gentian violet solution is made by adding one tablet of gentian violet to two cubic centimetres of ethyl alcohol and two cubic centimetres of distilled water.

The eye is cocainized by means of a 2% solution of cocaine. As little cocaine as possible is used, as it makes the cornea cloudy and inhibits further observations. Assuming the hole to be in the region of "four o'clock," a mark is made at four o'clock and ten o'clock on the limbus with the gentian violet solution. These two marks and the hole should appear in the same straight line. If they do not, the two marks are adjusted and further observations are taken with the ophthalmoscope. Finally, when a satisfactory position has been found for these marks (and the surgeon cannot be too particular regarding them), a little solid indian ink is mixed into a paste with water and the marks permanently tattooed at the limbus (Figure II).

This procedure of marking the hole at the limbus is rather an ordeal for the patient, so that it is usual to accomplish it on the day previous to operation.

The Operation.

Special Instruments.

The special instruments required for the operation are: (i) Steel dividers, (ii) steel millimetre rule, (iii) Goulden's retractor, (iv) Paquelin cautery with a number 13 point or an electric cautery with a bulbous platinum point. All these instruments are obtainable at Messrs. John Weiss and Sons, of Oxford Street, London.

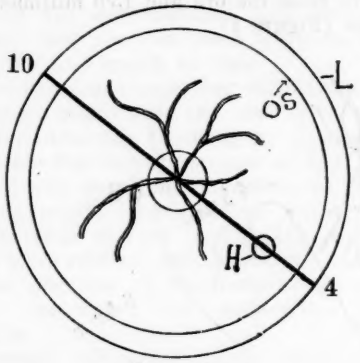

FIGURE II.

The Technique of the Operation.

Two cubic centimetres of a mixture of equal parts of "Novocain" solution and one in 5,000 adrenalin chloride solution are injected beneath Tenon's capsule over the site of the hole. The injection must be beneath the capsule as a subconjunctival injection does not sufficiently desensitize the sclera. After a wait of five minutes, dissect a flap of conjunctiva from one-third of the circumference of the limbus towards the fornix, so as to expose the sclera over the site of the hole. Then at ten o'clock (still assuming the hole to be in the ten o'clock-four o'clock meridian) insert a number 1 silk suture into the conjunctiva and carry it across the anterior surface of the cornea to the four o'clock mark and onwards, in the same meridian, round the exposed sclera. Thread the suture through the central perforation of the Goulden retractor, which keeps this guiding suture in position and retracts the conjunctival flap at the same time. With the steel dividers mark off a distance of thirteen millimetres along the guiding suture from the limbal mark at four o'clock. The posterior point of the dividers should then be directly over the retinal hole. Mark this spot permanently with a little indian ink paste (Figure III).

Now insert into the conjunctival flap two number 0 silk sutures, so that the flap can be tied into position immediately the puncture has been performed. The cautery itself should next be tested. The Paquelin cautery gives undoubtedly the best scar, but its behaviour is so fickle that I now recommend an electric cautery with a bulbous platinum point. Not until the cautery glows at white heat should the operation be proceeded with further, for the cautery, when being withdrawn from the puncture, will stick to the sclera unless it is at white heat.

Now incise the sclera over the 13 millimetre point with a sharp von Graefe knife. The incision should extend about two millimetres on either side of the 13 millimetre mark and should run in the line of the ten o'clock-four o'clock meridian (Figure III). Let out all the subretinal fluid, but do not go deeply enough with the von Graefe knife to injure the retina itself, or a further hole may be made in it. Then plunge in the cautery point at white heat

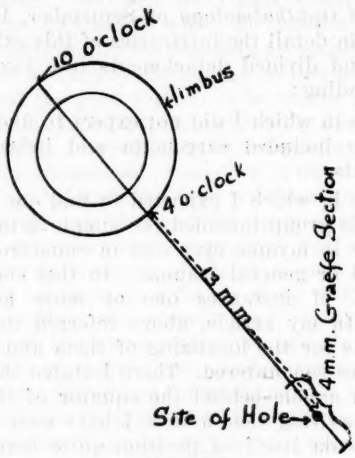

FIGURE III.

(the assistant should have it ready at such a temperature) and leave it in for from three to five seconds. Remove it slowly and immediately tighten and tie the silk conjunctival sutures to prevent the escape of vitreous. Remove the number 1 silk guiding suture and double bandage the eyes.

Post-Operative Treatment.

Both eyes must be kept bandaged for seven days after the operation and the patient must be placed in such a position in bed that the hole lies in the most dependent portion of the retina. For example, if the hole is at four o'clock, the patient lies on the left side and is kept rigidly in that position while the bandages are *in situ*. The first dressing is done on the fourth day and each day thereafter. A drop of a 1% solution of atropine is instilled into the affected eye at each dressing. A single bandage only is used after the seventh day. The patient is then allowed to rest quietly on his back. If, on the tenth day, there is no sign of vitreous hæmorrhage, the patient is allowed up. Several vitreous hæmorrhages have been seen by me. Some of them have had disastrous consequences. Gonin advises a hypodermic injection of "Coagulin" (Ciba) half an hour before operation, but I think this preoperative treatment is expensive and ineffective.

If it is found at the end of two weeks that the hole is unsealed and the retina still detached, a further attempt must be made at once to close the hole. Likewise, when multiple holes are found at the first examination and all cannot be closed at one sitting, a second operation may be attempted

from two to three weeks after the first. But whenever a second operation is performed, great care should be taken not to interfere with the previous scleral scar or vitreous will be lost and the globe will collapse.

Success is best proven by mapping the visual fields once before operation and afterwards at regular intervals over a lengthy period. Only when the field has remained full for more than a year can the surgeon claim complete success.

Central vision is often not materially improved if the macula has been involved in the detachment, but this is of minor importance compared with the restoration of full visual field and the prevention of complete blindness from progressive detachment of the retina.

Summary.

1. In simple detachments of the retina there are to be found three main types of hole, namely: Round punched-out holes, horseshoe shaped rents, and disinsertions at the *ora serrata*.

2. The hole or holes found must be localized and accurately marked at the limbus previous to operation.

3. In order to reattach the retina permanently, the hole or holes must be completely occluded by the point of a white hot cautery passed through the sclera over the site of the hole.

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Reports of Cases.

TWO ABDOMINAL TUMOURS.

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THE following two cases show the difficulties sometimes met with in abdominal diagnosis.

Mixed Neoplasm of Right Ovary.

Mrs. W.B., aged fifty-six, presented herself recently for treatment of a swelling in the right iliac fossa associated with pain, occasional abdominal cramp and gradually increasing constipation. She gave a history that some two years before, in a country centre, she had had an operation for a tumour of the colon on the left side. Examination showed her to be a very well nourished woman of good colour. There were two scars on the abdomen, one over the right *rectus abdominis* and a large one over the left rectus. Her husband explained that the first operation, which had been for right-sided abdominal discomfort with progressive constipation and occasional attacks of blood-stained diarrhœa, had shown that the suspected appendiceal trouble was really a neoplasm of the pelvic colon. A resection of the carcinoma was then done through the left rectus incision and end-to-end anastomosis performed. The suture line leaked and at a later date the fistula was successfully closed. All went well for over a year, when the present symptoms came on. Abdominal palpation revealed a large tender mass in the right iliac fossa which was provisionally diagnosed as a tumour of the ilio-caecal junction. A barium X ray meal did not support this idea, but a barium enema showed a relative narrowing, but no clear-cut stricture of the pelvic colon. The abdomen was then opened through the right rectus and as practically no free peritoneal cavity could be found, owing to plastic adhesions, exploration proved very difficult. However, it was revealed that the large right-sided tumour was a solid neoplasm of the right ovary which had risen from the pelvis and extended to the inferior aspect of the ileo-caecal junction. Examination of the pelvic colon showed that the carcinoma had begun to recur in the peritoneal coat of the bowel first and that cancer cells had become displaced into the pouch of Douglas and had first infected the peritoneum there and subsequently invaded the ovary, so that a mixed cell transplantation—cancer showing columnar carcinoma cells had arisen in that organ—an unusual type of ovarian new growth. No palpable secondary deposit could be discovered in the liver. As the ovarian tumour was hopelessly fixed in the pelvic floor; and as the colonic growth had recurred, no more could be done. The abdomen was closed. Considerable alleviation of the pain and abdominal discomfort has since resulted from treatment by deep therapy. Later on, a transverse colostomy may be called for as a palliative measure.

Renal Cyst Simulating Pyloric Carcinoma.

G.D.R., a man, aged seventy-two years, had complained for some few weeks of epigastric discomfort, belching of large quantities of gas, occasional attacks of vomiting and of loss of weight amounting to about 5·75 kilograms (thirteen pounds) over a period of five months. Physical examination showed that he had a very dilated stomach with splashing sounds, but no pyloric mass could be palpated. He also had a right-sided, long-standing oblique inguinal hernia and his heart was fibrotic. There was no tenderness in the right iliac fossa and indefinite guarding and tenderness in the right hypochondrium. There was no evidence of movable kidney on either side. A provisional diagnosis of an early pyloric neoplasm was made. Barium meal X ray examination by Dr. McDowall revealed a large hypotonic stomach with irregularity in the pre-pyloric zone. The patient refused exploratory operation and asked for treatment along medical lines. For some weeks he improved, but this improvement not being maintained, he was admitted to the Mater Misericordiæ Public Hospital and a further X ray examination confirmed the findings of the previous one. He again declined operation and was treated for a time by his own medical attendant, again on medical lines. Finally, tiring of the non-success of this, he was again admitted to hospital. Laparotomy on November 17, 1930, through the right rectus sheath revealed a large hypotonic stomach with no evidence of a pyloric new growth or new spasm. Further examination, however, showed a fairly large cyst arising from the lower pole of the right kidney and pushing forward behind the duodenum and hepatic flexure of the colon. That this kidney occupied a very high position and was not movable accounted for my inability to palpate this cyst at previous examinations. As the left kidney felt normal, and as the urine was normal in amount and contained no albumin, pus or sugar, it was decided to remove the cystic kidney, which had evidently been causing the gastric symptoms by pressure and reflex spasm of the duodenum. The nephrectomy was quite easy. The patient did well for about twenty-four hours and a fair amount of urine was secreted by the remaining kidney. He then complained of severe right-sided abdominal pain, which was not due to any obstruction in the preexisting inguinal hernia. A reflex anuria developed and he died forty-two hours after operation with symptoms of cardiac insufficiency.

Obviously it would have been wiser to have had renal functional tests done after finding the cause of the gastric symptoms, instead of going straight ahead and doing the nephrectomy. My only excuse for doing the latter operation then and there was that the ureter was small and atrophic and little true renal cortex appeared to be present, which seemed to justify the belief that the other and palpably more normal kidney was really the functional one.

TRICHOMONAS VAGINALIS.

By H. G. Furnell, M.B., B.S. (Melbourne), F.R.C.S. (Edinburgh),

Surgeon in Charge of the Female Venereal Disease Clinic, The Alfred Hospital, Melbourne.

The note on the subject of *trichomonas vaginalis* by Dr. H. D. Lethbridge in the issue of February 21, 1931, has revived my interest. In recent years there have been several excellent articles in the *American Journal of Obstetrics and Gynecology* on this subject and for more than a year I have been making sporadic attempts to find trichomonas in likely subjects. Perhaps because of unsatisfactory methods I did not succeed, though in several cases I saw what were probably dead trichomonas.

Some two weeks ago Dr. Gandevia referred to me a patient with probable gonorrhœa, with the following history. The patient was aged twenty-two, she denied coitus. Two weeks previously she noticed a profuse and very irritating vaginal discharge. Dr. Gandevia had taken smears and a pathologist had reported the presence of pneumococci. I had the smears repeated and received the following report: Cervix, Gram-positive diplococci; fairly numerous pus cells, no gonococci; urethra, few endothelial cells, no organisms of note.

Clinically, the condition was by no means a typical gonorrhœa. The vulva was bathed in a free discharge of slightly greenish, milky pus. The vagina was intensely inflamed and swollen, this condition extending over the portio, where there were numerous spots of deep strawberry red colour; there was no apparent endocervicitis nor urethritis. Some of the pus was removed on the blade of the speculum, placed on a slide and diluted with warm saline solution. Under the high power, numerous very active trichomonads were found. They are about twice the size of a pus cell, pear-shaped or elongated, and have at one end four small flagellæ in extremely active motion. At the other end is a short, thick axostyle.

The trichomonads are best seen in the little lake-like spaces among the pus cells and they move rapidly in and out of the field. In the high power fields usually five or six were to be seen. Under the microscope they retained their activity for at least twenty minutes.

The following treatment has been commenced: (i) Thorough scrubbing of the vagina with a swab, using green soap and a little water. (ii) Drying of the vagina and painting with 2% mercurochrome. (iii) Swabbing with glycerine, leaving it *in situ*.

The *trichomonas vaginalis* was first described by Donné in 1837 and C. H. Davis quotes various observers as to the frequency with which it is found in persistent, abnormal vaginal secretions, varying from 28% to 69%. Brumpt found them in 10% of women examined in a gynæcological clinic in Paris.

The trichomonas has been cultured in broth media to which fresh human serum has been added and, working with cultures, Davis found that a temperature of 44° to 45° C. (113° F.) for ten minutes produced clumping and practically destroyed activity, whilst no growth could be obtained in cultures from these trichomonas. As a temperature of 114° F. can be obtained in the vagina by medical diathermy, without pain or damage, this may prove to be a useful method of treatment, which I hope to try when a suitable case presents itself.

Since writing the above I have found the trichomonas in another patient, Mrs. D., aged forty-four years, a *multipara*, who consulted me privately about three months ago with a history of gonorrhœa which had been treated for over two years. A smear revealed gonococci in the cervical discharge. She only came for treatment once a week and several times I have noticed a general vulvovaginitis which did not seem explainable by the rather small amount of cervical discharge. She complains of a great deal of irritation, especially after menstruation. On examination of a fresh specimen of her somewhat scanty vaginal discharge, the flagellate was found in moderate numbers, at least one to every high power field. In view of the fact that a morphologically identical trichomonas is found in the intestine, I have advised her, in addition to the ordinary treatment, to wash the anal region thoroughly with soap and water after each defæcation and to keep the perineum well powdered and dry.

Bibliography.

C. H. Davis: "Leucorrhœa with Special Reference to Trichomonas Vaginalis," *American Journal of Obstetrics and Gynecology*, Volume XVIII, August, 1929, page 196; "Trichomonas Vaginalis, Donné: Experimental and Clinical Observations," *ibidem*, Volume XVIII, October, 1929, page 575.

H. D. Furniss: "Trichomonas Vaginalis," *American Journal of Obstetrics and Gynecology*, Volume XX, July, 1930, page 93.

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TREATMENT WITH ANTIVENINE TWENTY-FOUR HOURS AFTER A BITE BY A TIGER SNAKE.

By H. T. Tisdall, M.B., B.S. (Melbourne),
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AND

J. E. Sewell, M.B., B.S. (Melbourne),
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(From the Walter and Eliza Hall Institute, Melbourne.)

The patient, F.S., was a man aged forty-three, weighing seventy-one kilograms. He was admitted to hospital twenty-four hours after being bitten twice in rapid succession (one fang only penetrating) on the dorsal surface of the middle finger of the right hand at 8.30 p.m. on March 17, 1931. He at once applied a ligature at the base of the finger and a second round the wrist, and having scarified both puncture marks, washed the surface with a solution of potassium permanganate. The ligatures remained in position for twenty to thirty minutes.

He vomited twice about midnight. There was pain in the affected arm, radiating to the axilla, and some headache. During the following day the headache continued; he vomited some seven or eight times and developed weakness of the lower limbs and unsteadiness of gait, blurring of vision and bilateral ptosis. He had difficulty in opening his mouth and in protruding his tongue.

When admitted, he was in a semi-stuporose condition, with obvious dysarthria (associated with inability to open the mouth or protrude the tongue). There was bilateral ptosis, the left lid being dropped rather more than the right. The pupils were widely dilated and completely inactive. There was striking loss of power and some incoordination of the skeletal muscles, which were very well developed. Apart from numbness in the right hand, there was no sensory loss nor any paræsthesia. There were no other neurological signs.

The urine contained some albumin, a few red blood corpuscles and many epithelial cells. There was no free hæmoglobin. The respirations were regular, twenty-four per minute, and the pulse 92 per minute and of good volume.

After a preliminary subcutaneous injection of one cubic centimetre of antiserum to ascertain whether he was excessively sensitive to horse serum, 90 cubic centimetres of antivenine (an amount capable of neutralizing 45 milli-

grammes of dry tiger snake venom) were injected intravenously, the injection being completed at 9.30 p.m. At 11.30 p.m. a further injection of 60 cubic centimetres of antivenine was given intramuscularly.

By 9.30 a.m. on March 19, 1931, the patient had exhibited a little improvement. There had been no further vomiting and the mental condition was brighter, but the signs and symptoms otherwise remained the same. By 10.30 a.m. the pupils had contracted down and were now found to react to light and accommodation. During the day his dysarthria became progressively less and he was able to take a little food.

On March 20, 1931, the ptosis was somewhat less. During the succeeding days his muscular power and coordination steadily improved and by March 27, 1931, ptosis was only just recognizable. There was still some weakness in the right hand, possibly due to the ligature. On the evening of March 24, 1931, there was an extensive serum rash with slight pyrexia which had cleared up by March 27, 1931.

Comment.

The general results of animal experiments, using other venoms and antivenines, suggest that antivenines are not likely to be of much value in saving life when administered more than twelve hours after a bite. While it is possible that in this case a dose of venom which was just sublethal, had been injected in two snap bites by a tiger snake three feet long, the condition of the patient on admission led us to believe that a fatal issue was almost inevitable. Had the antivenine been administered immediately after the bite, a much smaller dose would have been adequate to neutralize all the venom in the circulating blood and prevent the onset of any symptoms. Believing that most of the venom was fixed, a high concentration of antibody in the blood was induced by intravenous injection and maintained by the slowly absorbed intramuscular injection in the hope that a sufficient amount of the venom, fixed or in process of being fixed to the tissues, might be mobilized and neutralized.

Though this case affords no proof of the occurrence of such a change, the history is highly suggestive.

Acknowledgements.

Our thanks are due to Dr. Hume Turnbull for permission to place this case history on record, and to Dr. C. H. Kellaway, who saw the patient and outlined the treatment.

Reviews.

THERAPY.

CAMPBELL'S "Handbook of Therapeutics" is designed to fill a niche on the shelf of the student's library which has been vacant for a long time.[1] This little book is well written, well illustrated and is printed in good, readable type.

The introduction is a thoughtful and well balanced statement of the aims and ideals of therapeutics. A chapter on the management of the patient mentions the salient points of good nursing. In dealing with the prescription, small points at variance with usual convention are the allowance of ten cubic centimetres as the equivalent of a half ounce dose, and the writing of the vehicle in the genitive instead of the accusative case. "Modes of Administration of Drugs" is a very practical chapter. Then "Diet," "Hydrotherapy," "Biological Products" and "Physical Methods" are successive subjects of consideration. These sections are excellent, but all too brief. Also hydrotherapy usually is included under the heading of physiotherapy. Psychotherapy is not mentioned. Chemotherapy is not treated separately. The remaining three-quarters of the book comprise a pithy outline of the treatment of the various system diseases. This is in the tradition of the older text books of therapeutics, and provides the student with a ready summary of appropriate treatment. This systematic treatment is provided, of course, in text books of general medicine. The earlier part of the book is so well conceived and handled that it might well have been expanded to absorb under its respective therapeutic methods the more detailed information given in the later chapters. The author has produced a book which provides the student with a sound ground work for his therapeutic studies and it deserves to be popular.

[1] "Handbook of Therapeutics," by David Campbell, M.C., M.A., B.Sc., M.D.; 1930. Edinburgh: E. and E. Livingstone. Crown 8vo., pp. 430, with illustrations. Price: 12s. 6d. net.

PROGRESS IN OBSTETRICS AND GYNÆCOLOGY.

"OBSTETRICS AND GYNÆCOLOGY," of the 1929 "Practical Medicine Series," edited by De Lee, Greenhill and Polak, presents a review of the year's progress in obstetrics and gynæcology. It is a most useful book for both specialist and general practitioner.[1]

The method adopted is the presentation of a *précis* of the important papers of the year in English, for all countries, with a brief editorial comment.

In the obstetric section there are 348 pages and 320 papers and a few illustrations. In the gynæcological section there are 290 pages and 130 articles and numerous illustrations.

The editors have performed their task well. The work of condensing so many papers has been deftly done. The gist of every important article is there and, indeed, of some that might have been left unwritten without prejudice to the progress of science. It is in this latter case that the editorial judgement will be found good for those who are inclined to be blown about by every wind.

A SURVEY OF PSYCHOPATHOLOGY.

IN "Psychopathology" Dr. Ernest Nicole has made a survey of current doctrines, his object being, as he explains in the preface, to fill in the gaps between text books of psychology, psychopathology and psychiatry.[2] The teachings of various present day schools are outlined more or less impartially and some attempt has been made at harmonizing different conceptions. In an historical introduction there is a brief account of French psychopathology, including the views of Charcot, the Nancy school, Babinski and Janet. Janet, indeed, has received scanty notice and the impression is conveyed that his teachings are purely of historical interest. However, he is briefly referred to later in the book. Subsequent chapters are devoted to Morton Prince, Freud, Adler and Jung, whose works are described fairly adequately. In a chapter on the theories of Rivers, Dr. Nicole might usefully have traced back the neuro-biological conception of psychopathology to the teachings of Hughlings Jackson. In the same chapter Dr. Nicole refers to Dibblee's (the name is misspelt in several places) theories of instinct and intuition and to Kinnier Wilson's transcortical inhibition and control with a brevity which presupposes intimate knowledge on the part of the reader. Watson, Kempf and Berman have each been allotted a chapter. Biochemical and physiological contributions are dealt with in a section of nine pages. The diligent reader who is not well acquainted with biochemistry, will look in vain for references to the literature on the hæmoclastic crisis and to Burridge's recent work, published in the *Journal of Mental Science*, July, 1929, and January, 1930. Devine's "Recent Advances in Psychiatry" richly deserves a place in the bibliography of this section. After a chapter on Kretchmer's constitutional

[1] "Practical Medicine Series": Obstetrics, Edited by J. B. De Lee, A.M., M.D., and J. P. Greenhill, B.S., M.D., F.A.C.S. Gynæcology, Edited by J. O. Polak, M.D.; 1929. Chicago: The Year Book Publishers. Crown 8vo., pp. 660. Price: $2.50 net.

[2] "Psychopathology: A Survey of Modern Approaches," by J. Ernest Nicole, L.M.S.S.A., D.P.M.R.C.P. & S., with a Foreword by W. H. B. Stoddart, M.D., B.S., F.R.C.P.; 1930. London: Baillière, Tindall and Cox. Demy 8vo., pp. 215. Price: 10s. 6d. net.

approach Dr. Nicole has devoted four pages to a discussion on combined trends, but has attempted little more than emphasize the need for correlation between the different conceptions of psychopathology.

The second half of the book contains appendices dealing with the concept of the *Ego* in psychiatry, type psychology (with a clear exposition of Jung's elaborate scheme) and the herd instinct. These sections are of interest, as they reflect the author's own views, but they contain a good deal of recapitulation and repetition of the earlier contents. Dr. Nicole refers to Trigant Burrows's "primary subjective phase" in infantile development in which the attachment of the child to the mother is not so much sexual in the Freudian "Œdipus" sense as due to an inability to discriminate between different sources of nutrition. He might well have quoted the observation of that celebrated authority, *Punch*, to the effect that the infant has first of all to learn the difference not between *meum* and *tuum*, but between mummy and tummy.

The author has included a wide field in his survey, but the compact form in which the matter is presented, is likely to prove bewildering to any but advanced students. In fact, one is not inclined to agree with Stoddart's reference in a foreword to a "predigested" literary banquet. The work would have gained vastly in value from more ample and detailed treatment of the material and by the inclusion of references to articles and papers in the bibliography. The reader who has already had a good grounding in psychopathology should find this book most acceptable, especially in the revision of studies for higher examinations.

INFRA-RED RADIATION.

THE infra-red rays lie on the side of the spectrum beyond the red; thus they are included in the invisible light waves which have been termed "black light."

It is only recently that their healing properties, apart from other rays, have been investigated, and in his book, "The Therapeutic Uses of the Infra-Red Rays," Dr. W. Annandale Troup gives a concise account of these rays, their action on the tissues of the body, and the results which have been achieved by their use in medicine and surgery.[1] Some of the author's patients have benefited in a remarkable manner, and if Dr. Troup's claims for the rays are confirmed by others, this form of treatment is deserving of an important place in medicine.

The infra-red rays are long wave rays, extending from about 7,700 Ångström units to beyond 500,000 Ångström units. It is the shorter of these rays, between 7,700 and 24,000 Ångström units, which are employed in treatment. To the infra-red rays many of the various forms of light and heat treatment owe their efficacy. With the ultra-violet rays, for which they are mainly used, the carbon arc, tungsten arc and mercury arc lamps give off a substantial proportion of infra-red rays. The instrument used by Dr. Troup, a modification of the Arnold infra-red generator, is made to give off pure infra-red rays of about 8,500 Ångström units. The modification which was designed by the author, does not affect the quality of rays generated, but purposes to make it possible to appiy the infra-red rays to three aspects of a joint or limb at the one time. This is an advantage.

There is no doubt that infra-red rays are capable of benefiting many conditions, more particularly those of an inflammatory nature. They are also credited with the power of relieving pain. Dr. Troup has had excellent results in the treatment of minor disabilities, particularly sprains. If his claim to be able to cure any sprain in three days be confirmed by others, his contention that it would be to the advantage of every medical man to instal an infra-red generator in his surgery will be justified.

The author cites a wide range of conditions which he has himself treated or which have been treated by others with infra-red rays. The list probably requires sifting, but the author's results suggest that the rays are capable of effecting considerable good in many disorders and disabilities. Unfortunately the existing level of exchange will increase the cost of these infra-red generators and limit their use. But those who desire to use this remedy and know all that is credited to the infra-red rays, will find ample material in Dr. Troup's book.

In a foreword to the book Sir William Willcox writes: "Dr. Troup is to be very heartily congratulated on the production of a very helpful and valuable guide in the use of one of the most important remedial agents which can be put into the hands of the medical practitioner."

It is to be regretted that with the copy of this work sent for review was included a typed suggestion as to the method which might be used in bringing the book under the notice of the medical profession. It is doubtful who was to blame for this procedure which constitutes an error of taste and judgement. It is to be hoped that this procedure will not be repeated.

[1] "Therapeutic Uses of Infra-Red Rays," by W. A. Troup, M.C., M.B., Ch.B., with Foreword by Sir William Willcox; 1930. London: The Actinic Press, Limited. Demy 8vo., pp. 57, with illustrations. Price: 5s. 6d. net.

Notes on Books, Current Journals and New Appliances.

AN AUSTRALIAN TRAVELS ABROAD.

WHEN Miss Nina Murdoch, of Melbourne, went to Europe, she went in receptive mood. The result is "Seventh Heaven," which has reached its third edition.[1] Miss Murdoch calls her book "A Joyous Discovery of Europe" and dedicates it "To those who dream of Europe." In Australia there are many such. If they read this book they will discover how to get the most out of their journeyings. Even if the author is tuned up to concert pitch the whole time, her enthusiasm is not forced. She writes well and does not allow her reader's interest to flag as she takes him from one country to another. She begins with Italy, takes a peep at Switzerland, passes to Belgium and Holland and on to London. Then come France and Paris. Her description of art treasures and her capacity for making her reader enjoy with her all that she sees are perhaps the secret of her success. The book is illustrated by reproductions of photographs chosen with discrimination. Those who have travelled in Europe will find in this book something to recall them to their old pleasures, especially if they have any devotion to the arts; to those who have not travelled abroad this book will be an *apperatif*. We congratulate Miss Murdoch, who is an Australian, on her successful and charming work.

COLLECTED PAPERS OF THE BAKER INSTITUTE.

THE first volume of the collected papers of the Baker Institute is to hand. It is a handsome book of crown quarto size, containing reprints of thirty-eight papers and of two manuals on diabetes, already noted in these pages. Every one of the thirty-eight articles has, we are pleased to note, been published in Australian medical journals. In the preface to the volume the Director of the Institute, Dr. W. J. Penfold, states that the clinicians of the hospital (The Alfred Hospital) have been encouraged to come to the Institute to work out, with the aid of its scientific and technical staff, "clinical problems which appeared to be at once urgent and hopeful." The result is seen in some of the papers in the series. The staff of The Alfred Hospital has reason to be grateful to the founders of the Institute. There is no need to enumerate the papers in the volume; most of them have appeared in this journal. The Director may well be proud of this record of achievement.

[1] "Seventh Heaven, a Joyous Discovery of Europe," by Nina Murdoch; Third Edition; 1930. Australia: Angus and Robertson Limited. Demy 8vo., pp. 268, with illustrations. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, MAY 16, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE CANBERRA CANCER CONFERENCE.

WHEN the Commonwealth Government some years ago bought a quantity of radium, the medical profession of Australia expressed its entire satisfaction and looked forward hopefully to its use in the treatment of patients over as wide an area as possible. Such striking results had been reported from other parts of the world and claims of such magnitude had been made, claims which apparently could be substantiated, that medical practitioners, apart altogether from the treatment of patients, were jealous of Australian prestige in cancer investigation. The proceedings of the second cancer conference held recently at Canberra have shown that the hopes of a year or two ago are being realized. A short account of the conference was published in the issue of April 25, 1931; some of the more important of the clinical reports to the conference will be published in this journal at a later date. The conference was largely one of representatives of institutions at which Commonwealth radium is used. Just as it is true that he who pays the piper may call the tune, so the Commonwealth Government has a right to ask those who use its radium to give an account of their stewardship. The conference, however, was something more than this. It was an attempt to gain "unity of action and purpose," to take stock of progress and to place cancer treatment on a complete basis.

In the summary of the review by Dr. M. J. Holmes of the work in all the States, the statement is made that the most modern and successful methods of treatment have been brought within the reach of all in Australia who suffer from cancer. The use of radium is not always the most successful method of treating malignant disease; hence this statement must be interpreted as referring also to surgical operation, deep X ray therapy and so forth. This is a very satisfactory state of affairs and the statement must be read in conjunction with the expressed need for the establishment of consultative clinics where they do not already exist. There is need to impress on the public and sometimes indeed on medical practitioners as well, the fact that availability of radium does not mean necessarily that radium should be used. Just as the application of radium should be made only by an expert, so the determination to use radium should be made by persons who know what radium can do, who know what end results can be expected from surgical operation, who have had sufficient experience to justify their recommendation or refusal of deep X ray therapy, whose clinical sense is keen and whose judgement is mature. These qualities are but rarely possessed by one man; hence the establishment of consultative clinics. Of the need for these clinics where they do not exist, there can be no question. The provision of facilities for treatment and the organization of bands of experts to direct its application are but two sides of the triangle. The third side is represented by the apprehension of the patient while his disease is yet in its early stage. As the report of the conference stated; the whole problem resolves itself into one of early diagnosis. For this both patient and medical practitioner are responsible. The patient must present himself to his medical attendant before a diagnosis can be made. The task of the general practitioner, for it is he and not the specialist who is concerned, need not be further pursued at the moment. The patient must be taught what constitute the danger signals and must be brought to realize the evils of procrastination. The conference

recommended that those in control of cancer treatment in the different States should be asked to investigate the possibility of instituting a general educational campaign throughout the Commonwealth. Such a campaign should without question be undertaken, but its organization will not be easy. Ways and means may be discussed at a later date. It must suffice at present to add that it is high time the leaders of the profession realized that the public is not receiving anything like adequate instruction on matters of health. It should not be necessary to consider the "possibility" of undertaking an educational campaign. The machinery ought to be in existence.

Only one or two of the main points emerging from the Cancer Conference have been discussed. Opportunity of referring to other aspects will be taken on another occasion. The most encouraging statement in the report is that declaring the intention of the Commonwealth Department of Health to act upon the several resolutions of the conference. The department will have the whole-hearted support of the medical profession in its fight against cancer.

Current Comment.

IMMUNITY TO PROTOZOAL DISEASES.

"WHY does man recover from disease?" This must have been one of the earliest questions asked by thoughtful students of medicine in the dim past. Later a knowledge of infection evoked the question: "Why do some men contract a specific infection to which they are exposed while other men remain immune?" A vast amount of work has been expended on the investigation of immunity, but complete answers to these questions have not been obtained. Though most of the work in immunity has been in relation to bacterial infection, the protozoal diseases have also been subjected to a great deal of investigation. It is well known that acquired immunity from certain protozoal diseases occurs. One attack of East Coast fever of cattle, due to *Theileria parva*, confers permanent immunity; rats recovered from infection with *Trypanosoma lewisi* cannot be reinfected; persons recovered from oriental sore, due to invasion by *Leishmania tropica*, are immune to further attacks by this organism. In fact, it is the practice in some places where the disease is common, deliberately to introduce infection on the arm, for example, in order to avoid the occurrence of the sore on the fact or neck, where it would be unsightly. Natural immunity is known to exist. Man is naturally immune to the effects of many of the protozoa of other animals, and some people also are apparently immune to infection by certain protozoa of man. The individual who, in a highly endemic area, escapes malarial infection, is known to every tropical medical practitioner. Very often this natural immunity should not be so-called, for it is rather a natural tolerance; protozoa are often present in the body, but are unable to cause any apparent disturbance of function. There has been much speculation concerning the cause of natural immunity, and a great deal of interesting experimental work has been done. Laveran showed that the blood serum of the baboon, which is usually immune to infection with *Trypanosoma gambiense*, when injected into an infected mouse causes a disappearance of parasites from the mouse's blood and may also be used successfully to protect mice against infection with *Trypanosoma gambiense*. But there are many factors to be taken into account when considering immunity from protozoal diseases; not the least of these is the degree of virulence of the infecting organism. It has been shown, for example, that the rapid passage of *Trypanosoma lewisi* through rats renders this parasite capable of infecting mice and guinea-pigs which are usually immune; it has been suggested that in some similar manner *Trypanosoma brucei*, the trypanosome of animals in Africa, may become infective to man, and, indeed, many claim that it is identical with *Trypanosoma rhodesiense*, the causal organism of Rhodesian sleeping sickness.

In his presidential address delivered before the Tropical Diseases and Parasitology Section of the Royal Society of Medicine in December of last year, Professor J. Gordon Thomson dealt with the question of immunity in man to protozoal diseases, especially to malaria, leishmaniasis, trypanosomiasis and amœbiasis.[1] He discusses immunity and virulence generally in introducing his subject, then proceeds to a consideration of immunity in these four most important protozoal diseases.

The introduction of the use of malaria as a therapeutic measure has provided much material for experimentation on immunity. Numerous investigators have noted that some individuals are definitely resistant to malaria; for example, S. P. James was unable to induce malarial paroxysms in 52 of 221 patients who received infective inoculations. James noted further that, though in some instances inoculation did not result in any clinical manifestations within the usual incubation period, malarial paroxysms occurred perhaps some months later. This applies only to benign tertian malaria, but Thomson remarks that he has found parasites of malignant malaria in the blood of Europeans who were not aware that they had been infected. He suggested that from this evidence it may be concluded that complete resistance or absolute immunity to invasion by malarial parasites is a matter of doubt. With regard to acquired immunity

[1] *Proceedings of the Royal Society of Medicine*, February, 1931.

he points out that Europeans suffer most acutely and frequently from malarial paroxysms during the early years of their residence in a malarious country. In hyperendemic areas almost all native children become infected and as a consequence their spleens become enlarged; as they grow older they acquire an immunity or tolerance to the plasmodia and the splenic enlargement tends to subside, so that among adults the enlarged spleen rate may be perhaps 2% as against a rate of 40%, 50% or higher among young children. Christophers was able to show that the incidence of splenic enlargement in hyperendemic areas was in indirect ratio to increasing age from childhood to manhood and in direct ratio to the average number of parasites found in the peripheral blood of individuals and the frequency of paroxysms. Other observers have noted that some people who have recovered from an attack of therapeutic malaria cannot be reinfected. Interesting experiments in passive immunity have been conducted by Sotiriades and Kauders, but their results cannot be taken as conclusive.

Natural immunity in man against trypanosomiasis was the basis of a dramatic experiment by Taute and Huber, who inoculated themselves and 129 African natives with blood containing *Trypanosoma rhodesiense*, with the result that not one developed trypanosomiasis, though numerous laboratory animals inoculated at the same time became affected with the disease. Yorke, Adams and Murgatroyd have shown that *in vitro* normal human serum rapidly destroys *Trypanosoma rhodesiense*, *Trypanosoma equiperdum* and *Trypanosoma congolense*, while *Trypanosoma gambiense* is apparently unaffected; they suggest, therefore, that man's immunity to the effects of trypanosomes of stock and "his relative susceptibility to *Trypanosoma gambiense* are bound up with the property of his serum."

Thomson remarks that there is no better example of acquired immunity to an animal parasite than the resistance of the game animals of Africa to trypanosomes; this depends on continuous inoculation by means of the tse-tse flies. Partial immunity in mankind may be observed in certain areas where natives are chronically affected with trypanosomiasis, of which the only evidence is enlarged cervical glands.

Some individuals have a tolerance to infection with *Leishmania donovani* similar to that possessed by some people for malarial infection. Napier and Das Gupta found that some people affected with post-kala-azar dermal leishmaniasis had previously suffered from a very mild fever only, and others again gave no history of any previous febrile disturbance at all. Many interesting experiments in passive immunity and diagnosis by means of skin tests, complement fixation tests *et cetera* have been carried out.

Walker and Sellards fed twenty men with cysts and free-living forms of *Entamœba histolytica*. Eighteen acquired infections, four only became affected with acute dysentery, fourteen became carriers, and in two no evidence of infection could be found. Thomson draws attention to the possibility that the pathogenicity or otherwise of a certain strain of *Entamœba histolytica* may depend on the type of intestinal bacteria present.

Partial immunity to amœbiasis is only evidenced by the existence of a low grade infection which does not cause clinically recognizable dysentery.

In summing up, Thomson mentions, among other things, that acquired immunity seems to depend on continuous infection and that if continued inoculation be interrupted for a period, the immunity breaks down; furthermore, debilitating conditions may result in a collapse of resistance.

It may be concluded that immunity to protozoal infections cannot be regarded entirely in the same light as immunity to the attacks of bacteria. A great deal of experimental work on this problem remains to be done. No one can conjecture whither a pursuit of this fascinating study may lead nor what great scope there may be in the way of effecting active or passive immunization against malaria and other protozoal diseases.

HERMAPHRODITISM.

A TRUE hermaphrodite should have every organ of both sexes. True hermaphrodites are very seldom found. At a recent meeting of the Royal Society of Medicine,[1] R. Rutherford reported a child, aged two years, whom he regarded as coming under this category. The child was brought for treatment of an inguinal hernia. The penis was 2·5 centimetres long. The scrotum had definite rugæ, but no testes could be felt. When the hernial sac was opened, a uterus, two Fallopian ducts and two sex glands were found. The relations of these structures to one another and their appearances were those usually found in a child two years of age. On microscopical examination testicular tissue and Fallopian tube tissue were found in one block; the *tunica albuginea* was invaded by cords of cells among which were large cells having the appearance of primordial ova with clear cytoplasm and a well defined nucleus. The uterus ended in lax cellular tissue. It is interesting to note that, though the male organs predominated, the child had the appearance of a female. Since microscopical examination revealed the presence of both types of sex organ, the child must be regarded as a true hermaphrodite. The mother was advised to bring the child up as a boy. The opinion was expressed in the discussion that the child might become a father, though the seminiferous tubules were not canalized. Rutherford said that if the ovarian element predominated the uterus might menstruate; he did not think it justifiable to interfere with the uterus until the child arrived at the age of puberty and showed signs of menstruation. He suspected that the *vas deferens* ended blindly.

[1] *Proceedings of the Royal Society of Medicine*, December, 1930.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Primary Carcinoma of the Fallopian Tubes.

RAYMOND E. WATKINS AND WILLIAM M. WILSON (*Surgery, Gynecology and Obstetrics*, July, 1930) have reviewed the literature of primary carcinoma of the Fallopian tubes. There are over 200 authentic cases on record. The majority have occurred at the time of the menopause. The actual cause is unknown, but in most instances long standing pelvic inflammation was a preexisting condition. In many instances other tumours existed in the ovary and uterus. The condition is found on microscopical examination to appear as one of two main types, either papillary or papillary-alveolar. There is a good deal of round cell infiltration of the mucosa and submucosa layers. There are no characteristic symptoms. Vaginal discharge, abdominal pain and menstrual irregularities occur in most cases. The discharge in the majority is watery and clear or straw coloured at first, later, after several weeks, becoming blood tinged. The discharge may be continuous or intermittent. There are no physical signs sufficiently characteristic to differentiate tubal malignant disease from adnexal disease in general. In most cases a tense cystic or solid mass was felt on one side of the uterus. The diagnosis is extremely difficult before operation. At operation the condition has frequently been taken for chronic salpingitis. The prognosis is very poor. Only six patients have survived the third post-operative year without a recurrence.

Basal Metabolism and Fibroids.

A. M. AGARONOW (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1931) has investigated the variations in the basal metabolic rate in cases of fibromyomata of the uterus. The majority of patients under forty years of age showed an increase, while the reverse was noted with those in patients over this age. If an increase was observed in the latter group, it was associated with degeneration of the fibroid and also climacteric symptoms. These changes in the basal metabolic rate were not due to the fibroids, but were caused by the endocrine glands. As his observations have been corroborated, the author considers that the development of fibroids is mainly influenced by hyperfunction of the glands of internal secretion, especially the ovary and pituitary.

Age Changes in the Cervix Uteri.

N. FREEDMAN (*American Journal of Obstetrics and Gynecology*, January, 1931) traces the epithelial changes from the first months of intrauterine life to old age. In the first few months there is no differentiation in the lining between uterus and vagina. About the fourth or fifth month the vaginal epithelium becomes differentiated into squamous epithelium and this also covers the lower part of the cervix. Then from the sixth month on the cervix begins to assume its characteristic mucous epithelial lining and pushes out the invading squamous epithelium to the external os. It may not stop there, but may continue and give rise to an erosion; on the contrary, it may not come down as far as usual. In the adult the cervix is subject to trauma in the form of lacerations at childbirth and these in a large majority become infected, erosions being produced. As already mentioned, even in the normal there is no fixed union of the two types of epithelium at the external os, but one can overlap the other, and in erosions this is greatly exaggerated. There is a strong inclination for the hyperblastic squamous epithelium to grow downwards into the stroma. The cervix is composed of restless tissue which is exceptionally liable to atypical growth due to the presence of two interchangeable types of epithelium. In making a plea for the prevention of cancer of the cervix the author advises that all should view the cervix as Charleton, who writes: "Let every chronic infected cervix be appreciated, not as a cervical catarrh, nor as an hypertrophy nor as a laceration, but as the prologue of an epithelial drama whose curtain may be malignant death."

Methods of Contraception.

H. ALBRECHT (*Münchener Medizinische Wochenschrift*, February 27, 1931), after discussing the medical indications for contraception, reviews the various methods usually advocated. He favours the use of the sheath, preferably associated with that of antispermatic substances by the female. When the sheath is refused, the safest method is to employ a rubber occlusive diaphragm of the Rameses type. This should be smeared with a jelly composed of lactic acid one part, boric acid one part and glycerine to 100 parts. Equally good results follow the use of a cervical cap, although this is more difficult to apply. In all cases the cap should not remain in position for more than twenty-four hours.

Thrombosis and Embolism.

G. SCHMIDT (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1931) reviews the cases of thrombosis and embolism occurring in the Breslau Clinic during the past ten years. Post-operative thrombosis occurred in 2·19% of the 6,114 operations and embolism in 0·05% of the same series. Intravenous injections, senility, arteriosclerosis and renal disease were not predisposing factors. Although ether anæsthesia is supposed to be a factor, especially of thrombosis, this was not borne out in the series. The majority of cases were noted in hard working peasant women who frequently presented marked degrees of visceroptosis, prolapse, rectal diastasis and varicosities. Thrombosis following delivery represents about 1% while embolism was noted in 0·05% of the labours. *Multiparæ* were more affected than *primiparæ*. Predisposing causes were severe hæmorrhage, either *ante* or *post partum*, subinvolution and operative interference, especially high forceps, version or manual removal of the placenta. Prophylaxis is mainly concerned with regular exercises, after operation or during the puerperium. They are contraindicated only when thrombosis already exists, with patients suffering from fever or after extensive operations or severe hæmorrhage. They should be started at once, for delay until the fifth or sixth day is liable to be associated with an increased risk of thrombosis or embolism. In three cases of embolism the Trendelenburg operation was performed without success.

Treatment of Pruritus.

H. STOLZENBERG (*Deutsche Medizinische Wochenschrift*, February 20, 1931) discusses the various treatments advocated for pruritus of the anal and genital regions. Careful examination must be made to exclude the numerous conditions which often cause intense pruritus, although in many instances no local or general cause can be found. The remedies suggested are legion. Radiotherapy is useful, but often fails. The author has had excellent results following the use of strontium preparations, either as an ointment or tablets for oral use. The details of two cases are given. Similar good results were noted in fifteen cases of subacute and chronic eczema, five of urticaria and one of *lichen planus*.

OBSTETRICS.

Icterus Neonatorum.

E. RONNENFELD (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1930) discusses the ætiology of *icterus neonatorum*—the severe type of jaundice, often fatal and tending to recur in successive members of a family. It is quite different from the transient physiological jaundice generally observed in most infants. This form is characterized by early onset, generally on the first day and rapidly becoming more intense. Meningeal symptoms are prominent, especially a characteristic cry or whimper. Hæmorrhage from the cord is common. The infant will generally take nourishment well. Death occurs after collapse. The spleen is enlarged, also the lymphatic glands, but no changes are noted in the liver. Petechial hæmorrhages and marked catarrhal changes in the mucous membranes, as well as staining of all serous surfaces, are common. In two cases observed by the author the only changes noted were icteric staining of the tissues and splenic enlargement, while the condition resembled a hæmolytic

jaundice. In a third instance there was a congenital stenosis of the bile ducts which other observers have not noted. Syphilis plays no part in the ætiology of the condition, nor does sepsis.

Placenta Prævia.

R. KESSLER (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1930) reviews the cases of *placenta prævia* in the Kiel Clinic during the past seven years. In a series of 107 (1% of the total deliveries) the maternal mortality was 4·7% and the infant mortality 43%. If the non-viable infants be excluded, the mortality drops to 25%. Treatment depends on the size of the os, the variety of *placenta prævia* present and the amount of hæmorrhage. In most cases of lateral or marginal *placenta prævia*, especially with *multiparæ*, the author advises either rupture of the membranes or bipolar version. However, if further hæmorrhage occurs, Cæsarean section is indicated. Section is always performed for central *placenta prævia* with non-dilatation of the os, whether the child be alive or dead. For the general practitioner bipolar version is the method of choice. While the use of a bag gives better fœtal results, hæmostasis is not so certain. Vaginal plugging is useless and leads only to a risk of infection. Finally, the author pleads for the immediate removal of all patients to hospital after the first attack of hæmorrhage.

A. H. BILL (*American Journal of Obstetrics and Gynecology*, February, 1931) makes a plea for more radical treatment of *placenta prævia*. When the placenta definitely overlaps any part of the os, the author considers only three procedures are worthy of mention: Cæsarean section, the use of a cervical bag and Braxton Hicks version. The maternal mortality rate is rarely lower than 6% or 7% and more often 10%. The fœtal mortality is very high. These methods, when hospital facilities are not available, are undoubtedly of great value. Ten authors in the last three years report a total of 2,117 patients treated by conservative means with an average maternal mortality rate of 9·68%. On the other hand, a group of four authors in the same period report 262 patients treated by Cæsarean section with a maternal mortality rate of 1·78%. The author points out that in *placenta prævia* death is usually the result of *post partum* hæmorrhage due to two distinct causes. One of these is injury to the cervix and lower uterine segment, due to extracting the child; the other is the general atony of the patient or shock resulting from the previous loss of blood and the effect of this condition upon the contractile powers of the uterine muscle. Anything which gives rise to stretching of the placental site, is apt to give rise to *post partum* hæmorrhage, even though there be no laceration of the cervix. The additional loss of blood in delivery is very likely to throw a patient who has had a previous loss, into a condition of shock, even though she does not seem to be in this condition before delivery. This further causes atony of the uterus. To obviate this condition the author advises prophylactic blood transfusion whenever the systolic blood pressure is below 100 millimetres of mercury and the red cell count below 3,000,000 per cubic millimetre. Transfusion should be given before, rather than afterwards as a last resort. The classical Cæsarean section is done to keep away from the placental site. A rectal rather than a vaginal examination is made to determine the condition. In conclusion, the author states that if there is little or no dilatation of the os, Cæsarean section should be performed whether the baby is alive or dead, for the viability of the child has no bearing on the choice of the method of delivery. If, however, there is considerable dilatation and the fœtal head is engaging, simple rupture of the membranes may control the hæmorrhage, or version may be performed if the head is high.

Post Partum Hæmorrhage.

K. HEIM (*Deutsche Medizinische Wochenschrift*, August 8, 1930) describes two cases of severe *post partum* hæmorrhage due to atony of the uterus following completion of delivery. In one instance the cervix was lacerated and repaired, but the hæmorrhage continued. Drugs, massage and packing the uterine cavity had no effect in either instance. Vaginal hysterectomy was performed, while blood transfusion was done after the main vessels had been secured. He reviews the possible lines of treatment in such cases of fulminant bleeding and does not consider blood transfusion as other than an adjunct to hysterectomy.

Nephritis in Pregnancy.

E. J. STIEGLITZ (*American Journal of Obstetrics and Gynecology*, January, 1931) holds that the term "low reserve kidney" has become perverted to mean a mild intoxication in pregnancy with slight changes, whereas, actually, a low renal reserve shows up much earlier and is associated with persistent and often excessive hypertension. He describes four groups: (*a*) simple albuminuria, (*b*) eclampsia and pre-eclampsia, (*c*) nephritis and hypertension in patients with preexisting renal disease, (*d*) nephritis and hypertension with complications, cardiac, syphilitic and otherwise. In an analysis of the patients 87% were found to be suffering from hypertension. This arises as a result of direct vascular irritation by the same factor which causes the renal lesion. In group (*a*), the syndrome of renal fatigue in pregnancy, the average systolic arterial tension was 153 and the average diastolic tension 97 millimetres of mercury. The average onset was at eight months. A reduction of renal reserve was shown by the inability of the kidney to secrete a urine of adequate specific gravity following deprivation of water for four to fourteen hours. The prognosis is good, both immediate and ultimate. In group (*b*) the onset was explosive in 92%; intoxication was more severe, hepatic involvement being found in this group whilst not in the others. The average arterial tension is higher, especially the diastolic (185 and 115 millimetres of mercury). A reduction of the renal ability to concentrate urine was found in 80%. In the group of nephritis in pregnancy with pre-existing vascular or renal disease it was found that the onset of symptoms was earlier—between five and six months. The arterial hypertension is present to a pronounced degree, an average of 194 millimetres of mercury systolic and 148 millimetres diastolic. Renal function was bad in 90%. The fœtal mortality rate averaged 60%. Among those followed up 80% were in a moderate or bad condition and each succeeding pregnancy leads to further renal degeneration. Cardiac inadequacy and severe anæmia are dangerous complications. Group (*d*) really overlaps and the symptoms are the same as the group to which they belong *plus* the added complications. The author is of the opinion that the renal tissues are injured synchronously with other tissues and that the hypertension is an expression of the action of the toxin on the vascular system. Great stress is laid on the inability of the kidney to concentrate the urine following water deprivation. From previous researches and clinical observations it is shown that alkaline administration to the point of alkalinization of the urine increases the albumin and is inadvisable. Diuresis is best induced with water and acid salts: calcium chloride or ammonium chloride. Œdema is essentially a tissue phenomenon and not necessarily renal in origin. The degree of hypertension is undoubtedly the most accurate clinical criterion. The relative hypertension is most important. A blood pressure of 150 millimetres of mercury systolic and 100 millimetres diastolic in a girl of nineteen may indicate a toxæmia from pregnancy *per se* as severe as that occurring in a woman of thirty-four with a systolic blood pressure of 190 and a diastolic pressure of 125 millimetres of mercury, if, prior to pregnancy, a vascular disease with hypertension existed. The author suggests the use of bismuth subnitrate in doses of 0·6 gramme (ten grains) three times a day to effect a gradual reduction of blood pressure. This may be deceptive, as it has no effect on the toxæmia and a false sense of security is given. The outlook for a succeeding normal pregnancy following eclampsia is less favourable than is generally believed. Chronic nephritis stirred up by pregnancy offers a poor future. Evaluation of the future of all classes should be based on the degree of anæmia, the degree of renal functional impairment, the age of the patient, and failure of recovery following delivery. The last is most important.

Special Articles on Diagnosis.

(Contributed by Request.)

XLV.

PROSTATIC OBSTRUCTION.

Of late years the science of urology has made a vast improvement in our knowledge concerning the condition of prostatic obstruction. Surgical interference, based upon a better recognition of the factors involved, is so successful and the mortality of this same surgery so diminished that an early and proper recognition of the condition becomes more and more obligatory, to the general practitioner especially. I say especially, as it is to him the sufferer applies in the first instance; the surgeon who treats such patients so often sees them only when the condition is well advanced and when some interference is only too obviously needed and often too late to be of any great value.

Prostatic obstruction occurs in the adult male any time after the age of forty years; at least, that is the earliest age in my experience. Roughly speaking, there are two types: (1) A median bar type, which is either glandular or inflammatory. (2) An adenomatous type, in which the median and lateral lobes are generally involved. Complications of this type are carcinoma, inflammation and calculus and also calculus in the bladder. The symptoms complained of are numerous and vary very much in different cases, but can be divided into two main heads: (i) Local or mechanical, (ii) constitutional or functional.

Local symptoms are difficulty, frequency and pain in urination and sometimes in defæcation. These can occur singly or in combination with each other. These local symptoms must of necessity be the first to show up and in consequence should be most carefully noted, as the constitutional ones are sequelæ and are due to the degeneration in the renal efficiency which must occur sooner or later, due to the prolonged and increasing interference with the proper functioning of the bladder. All types of renal infection are, of course, obvious complications, but more subtle complications are the dyspepsias, chest, heart and blood vessel conditions, and these are especially significant when they fail to respond to ordinary medical lines of treatment.

The advanced stage of prostatic obstruction is so obvious —recurrent attacks of retention, extremely difficult and painful frequency of urination, malaise, wasting, dry tongue *et cetera*, with a large mass felt *per rectum* and often another above the pubis—that there is no need in this respect to be more explicit. In such a case it is simply a question whether surgical interference is worth while or not; personally, I believe it is, from the patient's point of view, as the relief from pain is marvellous and such miraculous recoveries do take place, that nobody should be condemned to the ghastly end that is the lot of a patient with chronic prostatic obstruction, without at least an attempt to relieve suffering.

The earliest symptom of prostatic obstruction is frequency of urination, although in a few instances marked difficulty has been the only symptom complained of. The frequency is especially pronounced at night, and in the median bar cases, especially the inflammatory type, the history of frequency will generally date back many years and even as far as childhood. The frequency is sooner or later accompanied by signs of obstruction, the flow of urine is much diminished in size and force, there is difficulty in starting and finishing the act and the stream often stops and starts again. These symptoms are all grossly exaggerated if focal sepsis is present anywhere, and dental sepsis is a big factor in accelerating the production of the urinary infection which is bound to take place in any case of prostatic obstruction. When infection is present, pain on passing urine and often lack of control are present, both in starting the act and in finishing. Hæmorrhage can be severe, but is more commonly transient and often takes place with seminal emissions. All types of renal infection can occur, cystitis and prostatitis are common, and so is a mild type of epididymo-orchitis. Sooner or later complete obstruction occurs, especially if the patient happens to be caught in a position where he is unable to pass his urine when he has the desire to do so, or if he is exposed to cold, especially after taking alcohol. This retention may be only temporary and right itself, but often it needs the passage of a catheter and sometimes it is permanent and is then the indication for surgical operation that cannot be ignored. Even if the urinary function is restored, a careful examination will almost invariably show that it is not perfect and that a definite amount of residual urine is present, and once this is present, it tends to increase in amount, and once it increases beyond about four ounces, there is a definite increase in the rate of the patient's progress down hill.

Now nobody can look upon any surgical operation on the prostate as a trivial undertaking, and I would advise that the ideal time to choose for operation is when there is the slightest suggestion that the interference with urination is beginning to affect the renal function, or when the obstruction is without any doubt well developed, even without obvious renal changes. A careful examination of the patient will be suggestive about the former, as I mentioned under the constitutional symptoms. As regards the latter, several points will help to form one's opinion. First, watch the patient void his urine. One will often see the stream has no force at all and the patient has not noticed that it was so feeble because the change from normal had been so gradual. Then again it often dribbles only, stops and starts and so on. A rectal examination should then be done. An adenomatous enlargement of the lateral lobes will be easily detected, a uniform smooth enlargement on each side of a median groove, a groove which becomes gradually obliterated as the prostate enlarges. A median bar, glandular or otherwise, cannot be felt *per rectum*, and this type occurs more often before fifty-five years, the adenomatous type after.

Residual urine should then be tested. Now a metal instrument should never be passed in a man suspected of having prostatic trouble, unless there is present a very genuine likelihood of the need of surgical operation, and even a soft catheter should be used only when the patient is able to stay warmly and comfortably in bed afterwards. The risk of stirring up the whole trouble is a very real one. Therefore, to test for residual urine, get the patient to pass his urine when standing up and then let him lie down, pass a small catheter and see what amount of residual urine is present; stricture of the urethra and phimosis can be eliminated at this stage. I do not advocate a cystoscopy in every case, but it is essential to do it when a median bar is suspected, as it is the only way to make a positive diagnosis. Still more important, however, it is the only way to be sure that early lateral lobes are not present, and the presence of these is a definite indication for enucleation and not for a punch operation. Disorders of sex sensations occur frequently, but do not help in deciding on the need for interference or otherwise, especially in the early cases.

To sum up some of the more definite points:

1. Instruments, especially metal ones, should never be passed in a man with symptoms suggesting prostatic trouble, except with the greatest care, and never casually in the surgery.

2. A man over forty with acute retention ought to be seriously reviewed with a view to surgical interference, even if he regains apparently normal control.

3. If mechanical obstruction can definitely be proved, interference is well advised.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE CLINICAL MEDICINE SECTION OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Adelaide Hospital on December 17, 1930, DR. E. BRITTEN JONES, President, in the chair.

Lupus Erythematosus.

DR. W. C. T. UPTON showed a female patient, aged fifty-five years, who was suffering from *lupus erythematosus*. When the patient first came under observation the eruption was of ten years' duration. It was erythemato-squamous in nature and had the characteristic "butterfly" distribution on the face. After twelve injections of 0·1 gramme of "Solganol" given intravenously the eruption had almost faded.

Lupus Vulgaris.

Dr. Upton also showed a female patient, aged forty-three years, who was suffering from *lupus vulgaris*. The patient was born in South Australia. At the age of six, after an attack of measles, a small lesion appeared on the skin of the right cheek and gradually spread to involve practically the whole of the skin of the right side of the face. She had numerous forms of treatment, including scraping under general anæsthesia and frequent exposures to X rays. She was seen by Dr. Upton for the first time eighteen months before the meeting and at that time the affected areas manifested numerous "apple jelly" nodules under diascopic pressure. Ultra-violet light therapy was then used, generalized exposures with a mercury vapour lamp and massive doses being given locally with a Kromayer water cooled lamp. Dr. Upton pointed out that the condition had almost wholly cleared up and that the cosmetic result was good. He said that he intended to continue with this treatment.

Acute Labyrinthitis.

DR. R. G. BURNARD showed a man, aged twenty-five years, who was suffering from acute labyrinthitis. The patient was a clerk. His illness started suddenly on October 12, 1930, with giddiness and vomiting, but did not yield to the usual gastric sedatives. The patient was given relief by rectal injection of saline solution and glucose and by a hypodermic solution of morphine. At this time he complained that his left ear was sore and irritable, but neither then nor subsequently did he have any earache. There was some question of a slight discharge from the ear, but none was observed. On examination, the heart and lungs, the superficial and the deep reflexes and the urine were all found to be normal. Examination of the ear drum revealed injection of the vessels along the handle of the malleus, but no bulging of the membrane was present. Numerous shotty glands were palpable in the posterior triangle of the neck on the left side. When the patient got up during the next week he was still very giddy, and further examination revealed paralysis of the left side of the face. There was considerable loss of hearing in the ear and definite nystagmus was present, both when the vision was fixed and when the eyes moved to the left. On account of a definite history of tuberculosis in the family and because the patient had had hæmoptysis eighteen months previously, Dr. Burnard made a tentative diagnosis of tuberculoma involving the seventh and eighth nuclei, and asked Dr. A. L. Tostevin to see the patient. In the meantime a lumbar puncture was done; the cerebro-spinal fluid was normal.

A report from Dr. A. L. Tostevin was presented. Dr. Tostevin first saw the patient on October 31, 1930. At that time the temperature was 37·4° C. (99° F.). Paresis of the left side of the face was present and this was regarded as an intranucleous palsy. The vision of the right eye was approximately $^{6}/_{6}$ and that of the left $^{6}/_{9}$. Nystagmus was present when the patient looked to the left. The ocular fundi and the fields of vision were normal, but the examination caused the patient to retch and made him feel giddy. The hearing of the right ear, estimated with a watch, was $^{60}/_{60}$; that of the left ear was $^{12}/_{60}$. The right tympanic membrane was normal; the left membrane was slightly injected along the handle of the malleus, but otherwise it was normal. There was no discharge. Examination of the mastoid process by X rays revealed no abnormality. Cerebro-spinal fluid was normal. By November 4, 1930, the temperature had fallen to normal, the facial palsy was improving, the nystagmus was nearly gone and there was no vomiting as long as the patient kept quiet. On November 6, 1930, he was not quite so well, but the temperature was still subnormal. He felt giddy and vomited with the slightest movement of the head. There was no change in the condition of the ears, but the cervical glands were less swollen and the facial palsy was still improving. On November 25, 1930, the patient was able to get about and felt almost normal, although he had to be careful on making any quick movement. By December 10, 1930, he felt very well and had lost all feeling of giddiness. The hearing of the left ear was improving; it was $^{20}/_{60}$. There was no swelling of the glands of the neck, nor was there evidence of any facial palsy. Caloric stimulation of the left labyrinth still produced no response.

Addison's Disease.

DR. REX SARGENT showed a man, aged forty-three years. The patient was a returned soldier and unmarried. He complained of cough and increasing weakness over a period of two years and of severe pain in the right side of the chest for a week. The patient suffered from pneumonia in Egypt in 1916 and had been more or less debilitated since that time. He continued, however, to do fairly heavy manual work until two years before the meeting. At this time he began to notice that his weakness increased and that he became easily fatigued after very little exercise. Twelve months before, his friends began to remark on the increasing darkness of the skin of his face, neck, forearms and hands. He began to lose weight. In two years he lost 12·6 kilograms (two stone). He developed a persistent, dry, unproductive cough, accompanied by attacks of vomiting and of great weakness and dizziness.

On examination, the patient presented obvious signs of bodily wasting. Pronounced dark pigmentation was present in the buccal mucous membrane and the skin of the face, neck, upper part of the chest, areolæ of the nipples, umbilicus, scrotum and penis; there was no change in the axillæ. There were physical signs of bilateral apical pulmonary tuberculosis, which were consistent with the X ray findings. Dr. Sargent pointed out, however, that the sputum had been examined on many occasions and that no tubercle bacilli had been discovered. Signs of right side pleuritis were present. At the time of admission the systolic blood pressure was 110 and the diastolic pressure 75 millimetres of mercury.

Four months before the meeting it was observed that the liver was enlarged and that the patient had a decreased sugar tolerance. At the time of the meeting there was no abnormality in this regard and a section of the skin showed that there was a deposit of melanin which was confined to the basal cells. The blood sugar curve, though reaching 0·222 milligramme *per centum* one hour after ingestion of fifty grains of glucose, returned to normal in two hours.

Dr. Sargent explained that other causes of pigmentation, such as argyria and arsenical poisoning, had been investigated and had been excluded, and in his opinion the patient was suffering from Addison's disease. Dr. Sargent said that since admission to hospital the patient had become increasingly weak, had had frequent attacks of vomiting, and the blood pressure had fallen until at the time of demonstration the systolic pressure was 100 and the diastolic 65 millimetres of mercury. An X ray examination had been made for the purpose of discovering abnormality of the suprarenal bodies, such as calcification, but the report had not come to hand.

The patient was treated by absolute rest in bed. He was given light but nourishing diet and suprarenal extract was given in increasing doses; six grammes (ninety

grains) given every day produced no improvement. Dr. Sargent said that he reported the case because the three cardinal feature of the disease were exemplified, namely, extreme lassitude and weakness, the characteristic type and distribution of the pigmentation, and the very low blood pressure. All these were becoming more pronounced.[1]

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Crooke, Cyril Ednott, M.B., B.S., M.Agric.Sc., 1931 (Univ. Melbourne), St. Fillian P.O., Victoria.

Public Health.

NOTIFICATION OF BIRTHS IN VICTORIA.

THE attention of medical practitioners in Victoria is directed to the provisions of the *Births Notification Act*, 1930, which has been proclaimed as coming into force on April 1, 1931. The following memorandum has been received from the Public Health Department of Victoria:

The act applies to every municipal district in which there is an infant welfare centre under the control of and subsidized by the council and approved by the Minister of Public Health. It shall apply in the case of every child born alive or dead, and whether prematurely or at full time, but shall not apply in the case of the delivery of a non-viable fœtus.

It shall be the duty—

(*a*) of the midwife or (if no midwife is engaged) of any other person in attendance upon the mother at the time of or within twenty-four hours after the birth; or

(*b*) (where no midwife or other person is in attendance as aforesaid) of the father of the child if he is actually residing in the house where the birth takes place at the time of its occurrence; or

(*c*) (where the father is not residing in the house as aforesaid) of the occupier of such house; or

(*d*) (where the birth occurs in any public or private hospital or other public or private institution) of the matron, or other person in charge of such hospital or institution, or the person in charge of the portion of such hospital or institution in which the birth occurs—

to give notice in the form hereunder printed, or to the like effect, of the birth of the child to the municipal clerk of the municipality of the municipal district in which the mother of the child usually resides or, if such municipal district is not known to the person so giving notice, or is not a municipal district to which this act applies, then to the municipal clerk of the municipality of the municipal district in which birth occurs.

The notice shall be given by delivering the same at the offices of the municipality, or by posting a prepaid letter, letter-card, or post-card, addressed to the municipal clerk at the offices of the municipality, within forty-eight hours after the birth to which the notice relates.

On receipt of such notice the municipal clerk shall forthwith send the same, or a copy thereof, to the nurse whose duty it is to visit the house to which the notice relates.

Any person who fails to give notice of a birth in accordance with the act shall be liable to a penalty of not more than five pounds, unless the person responsible for notification can satisfy the court that he or she had reasonable grounds to believe that the notice had been duly given by some other person or that he or she had other reasonable grounds for omitting to give the notice.

[1] Since the meeting was held, the radiologist has reported the presence of calcification in the region of the right suprarenal gland.

Notification under the *Births Notification Act* shall be in addition to, and not in substitution for, registration under the *Registration of Births, Deaths and Marriages Act*, 1928.

Medical Practice.

COMMONWEALTH EMPLOYEES COMPENSATION ACT, 1930.

DR. J. G. HUNTER, the Medical Secretary of the New South Wales Branch of the British Medical Association, has forwarded the following statement:

The sections of the *Commonwealth Employees Compensation Act*, 1930, of most concern to medical practitioners are as follows.

Section 4.

"Employee" means any officer who is subject to the *Commonwealth Public Service Act* 1922-1930 and includes any temporary employee and any officer or employee to whom the Governor-General has declared that that Act shall not apply and any person who has entered into or works under a contract of service or apprenticeship with the Commonwealth, whether by way of manual labour, clerical work, or otherwise, and whether the contract is expressed or implied, is oral or in writing, but does not include (*a*) an outworker, or (*b*) any member of the Naval, Military or Air Forces of the Commonwealth.

Section 11.

In addition to any compensation payable by the Commonwealth under this Act to, or in respect of, an employee, the Commonwealth shall pay the cost, not exceeding in any case the sum of one hundred pounds, of such medical, surgical and hospital treatment in relation to the injury as is, in the opinion of the Commissioner, reasonably necessary.

Section 18.

(1) The Governor-General may appoint any duly qualified medical practitioners to be medical referees for the purposes of this Act.

(4) A medical referee who has been employed as a medical practitioner in connexion with any case by or on behalf of the Commonwealth or an employee, or by any insurers interested, shall not act as a medical referee in that case.

Section 19.

(1) Where notice has been given of an injury to an employee, or any employee is receiving weekly payments under this Act, any such employee shall, if so required by the Commissioner, submit himself for examination by a medical referee or a medical board consisting of two or more medical referees, and if he refuses to submit himself to such examination, or in any way obstructs the examination, his right to compensation shall be suspended until the examination has taken place.

(2) Where an employee is required by the Commissioner to submit himself for examination by a medical board, the employee may, within the prescribed period after being so required, nominate a medical practitioner who shall thereupon be one of the members of that board.

(3) An employee shall not be required to submit himself for medical examination otherwise than in accordance with this Act and the regulations or at more frequent intervals than are prescribed.

Regulations.

5. The fees payable to a medical referee shall be as follows:

For a first examination	£2	2	0
For a subsequent examination ..	£1	1	0

6. The medical referee, medical practitioner, or medical board to whom any matter is referred, shall give a certificate in accordance with Form C and shall forward

such certificate to the Commissioner or his delegate as soon as possible after the examination.

7. Where an employee has made a claim for compensation or is in receipt of weekly payments under the Act, he shall not be required, after a period of one month has elapsed from the date on which the first payment of compensation was made, to submit himself against his will for examination by a medical referee, a medical board, or a medical practitioner provided and paid by the Commonwealth, except as follows: At reasonable hours, once a week during the second month, and once a month during the third, fourth, fifth and sixth months after the date of the first payment, and thereafter once in every two months.

14. The authorities under the Commonwealth to employees of which the application of the Act shall extend shall be as follows: Commonwealth Bank of Australia, Commonwealth Savings Bank of Australia, Repatriation Commission, War Service Homes Commission, Commonwealth Railways, Australian Commonwealth Shipping Board, Council for Scientific and Industrial Research, North Australia Commission.

Obituary.

ANDREW BALFOUR.

DR. J. S. PURDY writes of the late Andrew Balfour as follows:

Sir Andrew Balfour, K.C.M.G., C.B., M.D., D.Sc., LL.D., F.R.C.P., D.P.H., Director of the London School of Hygiene and Tropical Medicine, died on January 30, 1931, at the age of fifty-seven.

The whole of the medical profession throughout the British Empire suffers a loss with the passing of this distinguished hygienist.

The members of the Australian Army Medical Corps who served in Egypt in 1915, will remember the excellent work done by the Sanitary Commission consisting of Sir George Buchanan, Sir Alexander Balfour, Sir William Hunter and Dr. Dudgeon, and especially the work of Colonel Andrew Balfour during his visits to Gallipoli and Lemnos.

The officers of the Third Australian General Hospital at Abbassia and of the Auxiliary Hospital at Luna Park will remember the large fly traps which Sir Andrew introduced.

It was my privilege not only to be associated with Colonel Balfour in Egypt during the world war, but from 1905 to 1907, when in the Egyptian Quarantine Service stationed at Port Said and El Tor in the Sinai Peninsula, I was in frequent communication with him. He was then Director of the Wellcome Research Laboratories at Khartoum and was carrying out the destruction of mosquitoes there on similar lines to those inaugurated by Dr. E. H. Ross, a younger brother of Sir Ronald Ross, who had already suggested the method successfully used by Dr. Pressat, of the Suez Canal Company, at Ismailia.

The work of the Wellcome Research Institute at Gordon College at Khartoum marked a new departure in research in tropical medicine and hygiene and has served as a model for similar work in other countries.

After the armistice Balfour took over the directorship of the Wellcome Bureau of Scientific Research in England and developed Henry Wellcome's pet hobby, the historical medical museum, which has been, together with the School of Hygiene museum, so ably organized and developed by Dr. Daukes.

On the establishment of the great School of Hygiene and Tropical Medicine in London in 1924, Balfour became its first director, and it is to his initiative, organizing capacity and skill in planning that the new school is recognized as possibly the finest institution of its kind in the world.

Australia is one of the few portions of the British Empire that Sir Andrew Balfour never visited, but in a letter to me in 1912 he had considered doing so. Evidently other interests intervened.

It is not only as an exponent of preventive medicine in general, and tropical hygiene in particular, that Balfour was so well and favourably known, but as the writer of "Lewis and Balfour's Public Health" he brought to bear a well trained literary mind. Such phrases as "the filthy feet of fæcal feeding flies" (one of his favourites) caught in the memory. In conversation he reminded one of Washington Irving's lines:

> For all he did, he had a reason,
> For all he said, a word in season,
> And ever ready was to quote
> Authorities for what he wrote.

As a sportsman, at rugby football he represented his university, Edinburgh, and won his international cap.

As a novelist, probably his best known work was "With Fire and Sword in the Soudan."

Notwithstanding his brilliant gifts and his great achievements, as a man he was modest and unassuming. As Dr. Charles Porter, a class fellow, now Editor of "Public Health," says: "He found his pleasures in his work, his books and an open air life when he could get it. He has died just when he might reasonably have been expected to have enjoyed the fruits of a life full of interests and of service to others."

Those who were privileged to have his friendship are the better for having known him and must mourn his passing as a great loss.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on May 4, 1931.

The Professor of Surgery reported the receipt for the Department of Surgery of the unconditional gift of his surgical library by Dr. Gordon Craig. The library comprises over a thousand volumes of classical, medical and surgical works, some rare and valuable and all extremely useful.

In addition to presenting his library, Dr. Craig has offered to the University valuable sets of medical instruments and the whole of the equipment of the Department of Urology. The total value of the gifts amounts to more than £1,200. The Senate has resolved to extend grateful thanks to the donor for his magnificent gifts and for his continued generosity to the University.

The Director-General of Health, as Chairman of the Advisory Committee of the School of Public Health and Tropical Medicine, reported to the Senate that material progress had been made by the school during its first year of existence.

Dr. L. B. Heath and Dr. W. I. T. Hotten were appointed Tutors in Anæsthetics at the Royal Prince Alfred Hospital for the current year.

The plans of the Medical School Rockefeller Foundation Building, as prepared by the Government Architect, were, on the recommendation of the Building Committee, approved. The estimated cost of this new building, which is to house several university departments, is £80,000.

The attention of the Senate was drawn to the fact that the jubilee of both the Medical School and the Engineering School would occur in the near future. The matter of celebrating the events adequately was left in the hands of the Vice-Chancellor and the deans of the respective faculties.

On the recommendation of the Faculty of Medicine, it was resolved that the hood for the degree of Master of Surgery should be of black silk lined with scarlet silk.

Correspondence.

PROFESSIONAL ADVERTISEMENT.

SIR: I have this day received a communication from our Honorary Secretary containing several important resolutions which were duly carried at an extraordinary general meeting of our association on March 19, 1931. I understand that there were less than fifty members present and that most of them were not general practitioners.

It has occurred to me that it is highly undesirable that resolutions affecting the whole profession intimately should be made law at a meeting at which it is impossible for the vast majority of the profession to be present. Country members pay the same subscriptions as city ones, despite their obvious inability to avail themselves of the same advantages, and one suggests that they should at least be allowed a say in their own control. To those who regard this as impracticable, I would suggest that there is such a thing as a ballot box. One ventures to hope that one or more country branches or even some of the suburban ones, will take the matter up.

In regard to the resolutions themselves, one understands that the meeting was by no means unanimous concerning their merits and demerits. And small wonder! We find: "(*a*) No member shall be a party to the appearance of his portrait in the public press." The rule is not qualified.

This resolution entirely ignores the fact that a medical man may have other interests than medicine. Politics, arts and literature all supply their quota of medical men, and it seems extraordinary that the public press should be forbidden to mention this side of their life. As an example, there is a federal election very shortly and unless something unforeseen happens, some paper or other will publish a photo of the leader of the Country Party. Such publicity is necessary from a political standpoint. I have no hesitation in stating my belief that our association would not dare to take any action against Dr. Earle Page for this distinct breach of By-Law 9, Section (*a*).

Again, does the association seriously contend that the late Sir Arthur Conan Doyle committed an infamous action in a professional respect when he gave the lay press a photo of the author of Sherlock Holmes? If one of our members should achieve the impossible and write a successful symphony, would we expel him if a newspaper obtained his photograph or even if the story of his life appeared in the "Musical Times"? If a second Caruso emerged from the medical school, would he be expected to go through life with a voice, but no face?

In regard to (*b*) it appears that the meeting contested it rather violently. It is a moot point as to whether we should extend to our patients the courtesy of informing them we have started work again. Without expressing an opinion either way, one feels that all our members should be allowed a vote on it.

Section (*c*), concerning indirect advertising, will be read, unfortunately with mild amusement, by anyone connected with journalism. Sporting papers publish full page signed articles on medical affairs by leaders in our profession, daily papers interview returning medical celebrities and near-celebrities, medical ministers for health and municipal health officers discuss health matters with passing journalists, named doctors give health talks over the air, and no action is taken. As a fact, the attitude of the profession in changing very quickly in the matter of endeavouring to educate the public in health matters. One understands even the King's physicians write signed medical bulletins. Only recently one noticed that Lord Moynihan gave a lecture on surgery "over the air" and *The British Medical Journal* gave him discreet praise for it. One also noticed that Australian surgeons invite the press to their annual meetings.

One feels that there is so much useful work to be done here in Australia in the way of offering the Government a sound scheme for: (i) The satisfactory governing of hospitals, (ii) the menace of the ever-increasing army of free out-patients, (iii) the education of the public on health matters (to mention only a few vital needs), that our Association is merely fiddling while Rome burns in its rather impudent attempts to tell doctors how to behave themselves.

I apologize for taking up so much of your space. My excuse is that I was not able to be present at the meeting concerned and express these same Bolshevistic views there.

Yours, etc.,

KEITH L. BARRY, M.B., Ch.M.

Leura,
New South Wales,
April 22, 1931.

Post scriptum. Would one call the publication of the names of our new office bearers in *The Sydney Morning Herald* an indirect advertisement?

THE ACTION OF CHLOROFORM.

SIR: I would like to send you the complete copies with electrocardiograms of my recent experiments in connexion with chloroform administration with and without the small preliminary dose of morphine and atropine, in which I am a very firm believer, as you know. I cannot, however, do this, at least, not at present, as I have only the original cardiograms, but I can send you copies of the remarks sent to me by Professor W. A. Osborne, Professor of Physiology in connexion with the Melbourne University, and also those of Dr. Hume Turnbull.

The experiments confirm what I have always stuck out for, that is, the "safety first" factor of a small dose of morphine and atropine in controlling the heart under anæsthetic induction and administration.

There is no question that chloroform is a dangerous drug if improperly handled. On the other hand, using it as I use it, every day and practically on every case (except in those in which I use open ethyl chloride) as a lead to soothing down the laryngeal reflexes prior to administration of open ether, it is a distinct blessing.

The mask of one layer of flannel should, however, never be brought down right in contact with the face. The administrator should start with the mask about an inch away from the face and then very gently bring it closer so that he finally has it about a quarter inch or less from the face.

In other words, chloroform must have free air.

I never use it as a means of full induction, but only as a means of leading to full induction. Of course, should I strike a case in which chloroform has to be given because of any risks connected with any other form of anæsthesia, I use it; but even in these cases, during the actual induction period I pour a few drops of ether upon the open mask during the early stages.

I think we have both ill-used and over-abused chloroform. I certainly believe it has great uses as a means to a pleasant and even induction if correctly handled and, in conjunction with the small one-eighth grain morphine and one one-hundred-and-fiftieth grain atropine, as a preliminary.

Vagal inhibition is the great risk during induction and this can be guarded against by morphine and atropine.

The experiments to which Professor Osborne and Dr. Turnbull refer took place at the Research Laboratory connected with the Melbourne Hospital on three separate dates: October 2, 1930, October 10, 1930, and November 19, 1930. Dr. E. W. Gault administered the chloroform. Miss Maudsley supervised the cardiograms and I was the subject.

October 2.—One administration sitting up and three administrations lying down. No preliminary of morphine and atropine.

October 10.—Administrating sitting up. No preliminary of morphine and atropine. It was on this day that the patient took a sudden turn during induction—went a blue-white colour and pulse dropped from 75 to 47.

November 19.—Preliminary one-eighth grain morphine and one one-hundred-and-fiftieth grain atropine. Administration sitting up in armchair. Pulse good throughout;

Report of Miss Maudsley.

Melbourne Hospital.

Dr. Hornabrook.

A. Without Atropine or Morphine.

(1) Lying down. When unconscious *T* is diminished in amplitude in all leads being inverted in III. *P* is slightly increased. The rate rises from 85 to 109 per minute.

(2) Sitting up. Patient tired. Soon after becoming unconscious a nodal rhythm is established, the rate falling from 75 to 47 per minute, subsequently before regaining consciousness normal rhythm returns with rate of 73, which rises to 86 with consciousness.

B. With Atropine and Morphine.

Sitting up. There is no change in the form of the curve when unconscious, the rate rises from 73 to 93 and falls to 62 with return of consciousness.

Report of Professor W. A. Osborne.

Dr. Hornabrook's experiment and the electrocardiograms obtained during the period when his circulation was faulty open up a new chapter, it appears to me, in the pharmacology of chloroform. Hitherto in dealing with the effects of this agent on the heart we have turned our attention chiefly to the direct poisoning action on the myocardium. When vagal excitement is thought of, it is invariably in terms of chronotropic change.

In Dr. Hornabrook's case the main cardiac disturbance was the change of site of origin of the systole from the normal sino-auricular node to the auriculo-ventricular node.

This means that the ventricular systole in both chambers was not preceded by an auricular systole. We are just beginning to understand from the dynamics of the heart how important the auricular prelude really is. Robbed of this preparatory action, the ventricular output is reduced and there is no compensatory quickening, but the reverse—or very obvious slowing. The sudden onset of the circulatory disturbance in Dr. Hornabrook and its quick relief, both of which I witnessed, would tend to the view that this nodal rhythm was of vagal origin. This is, I think, an entirely new idea and surely a very important fact in the study of chloroform anæsthesia. If the shifting of the site of origin of systole from the normal position is due to vagal action, then we are justified in assuming that this disturbance will be prevented by previous administration of atropine.

Report of Dr. H. Hume Turnbull.

I am returning your paper with thanks. It is, I think, very interesting and valuable. The electrocardiograms which are abnormal, are, I think, examples of alteration of pacemaker in the node, as Miss Maudsley suggests, and are probably vagal in origin, and not an indication of cardiac muscle weakness. This is suggested both by their type and the fact that they soon disappear in spite of continued administration of anæsthetic, which would hardly be the case if they were due to poisoning of muscle by the drug.

Report of Dr. R. W. Hornabrook.

The two outstanding points brought to light in connexion with these cardiograms are:

1. That it is not fair to the patient to administer an anæsthetic to him, especially chloroform, when he is tired. The risk is increased by so doing. This is common sense.

2. That the small preliminary dose of morphine and atropine before a general anæsthetic is a decided factor in lessening the risk and increasing the "safety first" factor, as well as assisting in reducing the amount of general anæsthetic required to maintain a patient in a stage of surgical anæsthesia.

Yours, etc.,

R. W. HORNABROOK.

120, Collins Street,
Melbourne,
April 28, 1931.

SUPERANNUATION SCHEME FOR DOCTORS.

SIR: The letter of Dr. Keith J. B. Davis on the above subject in THE MEDICAL JOURNAL OF AUSTRALIA of February 21 provides food for thought.

Financial stringency would probably prevent the satisfactory launching of such a scheme at present, but with the advent of more favourable times the idea should be popular.

Dr. Davis sees difficulties in the management. He suggests that arrangements might be made with some reputable life assurance society, and this certainly appears a satisfactory solution. Probably if one of the prominent assurance societies formulated a scheme and persuaded the local Branches of the British Medical Association to sponsor it, members of the profession would readily subscribe.

Yours, etc.,

A. R. SOUTHWOOD.

Adelaide,
April 28, 1931.

Hospitals.

ROYAL PRINCE ALFRED HOSPITAL.

FUNGI IMPERFECTI.

AT the first reunion of the Royal Prince Alfred Hospital Residents' and Ex-Residents' Association, held in October, 1930, the following statement on *Fungi imperfecti* was presented by the staff of the Department of Dermatology.

A lot of research work has lately been done on *Fungi imperfecti*, which are believed to be a connecting link between the true yeasts, that is, *Saccharomyces cerevisiæ*, first described by Tommasoli in 1889, and the ringworm fungi. The *Fungi imperfecti* are a lower order than the yeasts and reproduce by budding without ascus formation. Their classification is still incomplete, but they are said to belong to the *Thallophyta*. The monilias and pityrosporon groups are a lower order than the blastomyces and coccidioides which more closely approximate to the true yeasts. They consist of a vegetative part or mycelium of filaments or hyphæ, usually septate, and a reproductive part of spores which multiply by budding or sometimes develop from hyphæ as lateral buds or conidia. They are Gram-positive and grow on Sabouraud's maltose agar at 25° C. in twenty-four to forty-eight hours. Some are purely saprophytic, but others have been proved to be pathogenic in man. Among the latter are: *Monilia albicans*, *Monilia pinovi*, *Monilia candida*, certain cryptococci and blastomyces.

They have been recovered from numerous skin lesions, such as psoriasis, seborrhœa and allied conditions and themselves cause lesions formerly attributed to other causes, for example, perlèche, many intertrigenous lesions previously thought to be seborrhœa or the epidermophyta. They also frequently occur in lesions on the trunk. They are characterized clinically by a detached epidermal border enclosing a pinkish moist central patch, or may occur as minute vesicles. In some cases the infection may become generalized. Clinically they are indistinguishable from ringworm and may affect the nails and nail beds.

Thrush is another common condition caused by these organisms, particularly *Monilia albicans*. The coccidioides are the only members of the group to form asci and are the most closely allied to true yeasts. Perhaps the most interesting of the group is the pityrosporon, formerly known as the bottle bacillus, which was cultured in 1908 by Garner, of the Saint John's Hospital, London, and described by Dowling and McLeod in 1928. In scales it appears only as the old name implies, with the appearance of a budding yeast.

In culture on fluid media, mycelial non-septate threads are seen in abundance as delicate hyphæ with clubbed extremities and lateral buds, but no mycelium is seen in

solid media. Monilia, on the other hand, shows a scanty but thick mycelium in solid media, but none in a fluid medium. The pityrosporon has been proved to be the cause of the so-called seborrhœic lesions and has conformed to all Koch's postulates.

Post-Graduate Work.

COURSE OF LECTURES IN HOBART.

THE Organizing Committee for Post-Graduate Studies of the Tasmanian Branch of the British Medical Association has arranged to hold a course of lectures on May 21, 22, 23 and 24, 1931. The lectures will be given by Dr. Hume Turnbull and Dr. J. B. Colquhoun, of Melbourne. Dr. Turnbull will lecture on "Cardiac Infarction" and "Cardiac Problems in General Practice"; he will give clinical demonstrations on patients with cardiac conditions. Dr. Colquhoun will lecture on "Diseases of the Spine" and "Fractures"; he will give demonstrations on plaster work and the preparation of plaster of Paris splints. Further information may be obtained from Dr. W. L. Crowther, the Honorary Secretary, 190, Macquarie Street, Hobart.

Diary for the Month.

MAY 19.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 22.—Queensland Branch, B.M.A.: Council.
MAY 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 27.—Victorian Branch, B.M.A.: Council.
MAY 28.—South Australian Branch, B.M.A.: Branch.
MAY 28.—New South Wales Branch, B.M.A.: Branch.
JUNE 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 3.—Victorian Branch, B.M.A.: Branch.
JUNE 4.—South Australian Branch, B.M.A.: Council.
JUNE 5.—Queensland Branch, B.M.A.: Branch.
JUNE 9.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 11.—Victorian Branch, B.M.A.: Council.
JUNE 12.—Queensland Branch, B.M.A.: Council.
JUNE 16.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments.

Dr. S. Shineberg (B.M.A.) has been appointed Government Medical Officer at Bingara, New South Wales.

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Dr. V. H. Webster (B.M.A.) has been appointed Medical Officer of Health to the Wyndham Road Board, Western Australia.

* * *

Dr. F. H. U. Baker (B.M.A.) has been appointed Medical Officer of Health to the North Fremantle Municipal Council for the period of three months as from April 1, 1931.

* * *

Dr. W. Armstrong (B.M.A.) and Dr. D. Zacharin (B.M.A.) have been appointed Certifying Medical Practitioners at Violet Town and North Carlton respectively, pursuant to the provisions of the *Workers' Compensation Act*, 1928, Victoria.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

DIRECTOR-GENERAL OF PUBLIC HEALTH, SYDNEY, NEW SOUTH WALES: Honorary Medical Officers.
DOWERIN HOSPITAL COMMITTEE, WESTERN AUSTRALIA: Medical Officer.
THE WOMEN'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.
UNIVERSITY OF HONG KONG: Professor of Pathology.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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